

CHANGES IN ACOUSTIC REFLEX ACTIVITY
FOLLOWING TWO HOURS OF INDUSTRIAL NOISE
EXPOSURE IN NORMAL HEARING SUBJECTS

BY

GARY PHILIP RODRIGUEZ

A DISSERTATION PRESENTED TO THE GRADUATE SCHOOL
OF THE UNIVERSITY OF FLORIDA IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

UNIVERSITY OF FLORIDA

1986

ACKNOWLEDGMENTS

I would like to thank the faculty, staff and students of the Department of Speech for all their help. Their support and friendship made my stay at the University of Florida truly enjoyable. My sincere thanks go to Dr. Kenneth Gerhardt, chairman of my supervisory committee, whose knowledge, patience and guidance made this project possible. He embodied all of the qualities a student could possibly hope for, giving freely of his time and himself whenever called upon. I will always feel indebted to him, and hold his friendship in the highest regard.

I also wish to thank my supervisory committee, Drs. Alan Agresti, Alice Holmes, Joseph Kemker, and Patricia Kricos. Their expertise, advice, and encouragement were invaluable through my entire doctoral program.

Special thanks go to my parents, whose love and support remain a source of strength in my life, and my children, Casey and Rebecca, who helped keep everything in proper perspective, even during the roughest of times. My deepest thanks go to my wife, Diane, whose friendship and love provide meaning to all my endeavors.

TABLE OF CONTENTS

	<u>Page</u>
ACKNOWLEDGMENTS	ii
ABSTRACT	v
CHAPTER I BACKGROUND AND PURPOSE	1
Introduction	1
Review of the Literature	4
Temporary Threshold Shift	4
Physiology of the Acoustic Reflex	12
Theories on Middle Ear Muscle	
Function	16
Acoustic Immittance	22
Acoustic Reflex Threshold	33
Acoustic Reflex Magnitude	37
Temporal Characteristics of the	
Acoustic Reflex	39
Acoustic Reflex Adaptation	43
Acoustic Reflex Properties and	
Temporary Threshold Shift	51
Statement of Purpose	55
CHAPTER II METHODS	57
Subjects	59
Instrumentation and Procedures	59
Exposure	59
Behavioral Hearing Threshold	
Measurements	62
CHAPTER III RESULTS	86
Pre-Exposure Measures	86
Behavioral Thresholds	86
Acoustic Reflex Threshold	87
Acoustic Reflex Magnitude	90
Acoustic Reflex Latency	92
Acoustic Reflex Adaptation	97

CHAPTER III	(CONT.)	<u>Page</u>
	Effects of the Industrial Noise	
	Exposure	106
	Behavioral Threshold	106
	Acoustic Reflex Threshold	111
	Acoustic Reflex Magnitude	114
	Acoustic Reflex Latency	114
	Acoustic Reflex Adaptation	118
	Correlations	124
	Acoustic Reflex Threshold and	
	Reflex Threshold Shift	124
	Acoustic Reflex Magnitude	126
	Acoustic Reflex Latency	126
	Adaptation	129
CHAPTER IV	DISCUSSION	131
	Interpretation of Results: Pre-	
	Exposure Measures	131
	Acoustic Reflex Threshold	131
	Acoustic Reflex Magnitude	132
	Acoustic Reflex Latency	134
	Adaptation	139
	Effects of the Industrial Noise	
	Exposure	143
	Behavioral Threshold Shift	143
	Acoustic Reflex Threshold	146
	Acoustic Reflex Magnitude	147
	Acoustic Reflex Latency	149
	Adaptation	151
	Correlations	153
	Summary	155
REFERENCES		157
BIOGRAPHICAL SKETCH		169

Abstract of Dissertation Presented to the Graduate
School of the University of Florida in Partial
Fulfillment of the Requirements for the Degree of
Doctor of Philosophy

CHANGES IN ACOUSTIC REFLEX ACTIVITY
FOLLOWING TWO HOURS OF INDUSTRIAL NOISE
EXPOSURE IN NORMAL HEARING SUBJECTS

By

Gary Philip Rodriguez

August, 1986

Chairman: Kenneth J. Gerhardt, Ph.D.

Major Department: Speech

Research indicates that certain parameters of acoustic reflex (AR) function change following noise exposure, most notably, AR threshold. One dimension of AR function that has not been studied extensively is the relaxation or offset response of the acoustic reflex system. There is also a paucity of information regarding AR adaptation in response to industrial noise stimuli. Although continuous noise exposures may answer some of the questions regarding AR behavior, they cannot explain the role of the acoustic reflex system in an industrial environment.

This study evaluated changes in behavioral thresholds and AR activity following a two-hour industrial noise

exposure. Results show that significant threshold shift was obtained, along with changes in AR threshold, AR offset latency, and certain measures of AR adaptation. Potential relationships between various measures of acoustic reflex behavior, and expressions of temporary threshold shift (TTS) were also investigated. Findings indicate that AR magnitude and offset latency may be useful in explaining some of the variability typically encountered in TTS studies.

CHAPTER I BACKGROUND AND PURPOSE

Introduction

The acoustic reflex arc is a unique component of the auditory system because of its ability to process energy in several different forms. The middle ear acts as an impedance-matching device which facilitates the transformation of air-conducted disturbances into hydromechanical events. The cochlea then encodes this hydromechanical action into neural information which is sent to certain regions of the caudal brainstem. The brainstem nuclei through their efferent connections cause contraction of the middle ear muscles which results in an impedance change back at the tympanic membrane. As one might imagine the interaction of these various sub-components is quite complicated. Because of these complex relationships, however, the acoustic reflex arc provides an excellent opportunity to study several regions of the auditory system simultaneously.

Research indicates that abnormalities anywhere along the acoustic reflex (AR) system can cause a change in AR properties. If a conductive hearing loss is present,

acoustic reflex thresholds may be elevated or absent. Sensorineural hearing loss also results in a change in threshold and certain temporal characteristics of the acoustic reflex response. Retrocochlear pathology is known to delay or prevent the occurrence of certain AR events. Noise exposure has also been reported to cause changes in parameters of the acoustic reflex, namely magnitude and threshold. This is most likely due to the changes created within the cochlear partition, which serves to encode the acoustic signal for neural transmission.

Only limited information is currently available regarding the temporal properties of the acoustic reflex arc. The primary focus of recent research efforts has been in understanding the onset properties of the acoustic reflex response in normal and some pathologic populations. For example, it is well established that the onset properties of the AR are directly related to certain parameters of the eliciting stimulus. Intensity, duration, rise-time, and spectral content of the activating signal all have significant effects on the acoustic reflex response. Offset latency, on the other hand, shows little or no dependence on stimulus parameters. Because of this difference, some researchers suggest that the process of AR contraction and relaxation are inherently different and should be studied separately. It has also been suggested that AR offset latency may represent a closer estimate of neural conduction

time and, therefore, should be the focus of future research efforts.

Currently, data are available which suggest that AR temporal characteristics in subjects with sensori-neural hearing loss may differ from normal hearing populations. The etiology of these apparent delays is still unknown. Norris, Stelmachowicz, Bowling, and Taylor (1974) reported offset latencies of greater duration for a group of subjects with sensorineural hearing loss when compared to a group of normal hearing listeners. They theorized that differences observed between the two groups were caused by changes in cochlear function of the hearing-impaired subjects. In contrast, Borg (1976) showed longer AR offset latencies in animals with brainstem lesions. He suggested that the group of hearing-impaired subjects used in the previously mentioned study presented concomitant retrocochlear involvement. Even less evidence exists regarding the effects of noise-induced hearing loss on temporal properties of the AR. Temporary hearing loss in normal hearing subjects offers a unique model of cochlear stress, while controlling for possible contaminating effects of retrocochlear involvement. Such experiments may resolve some of the issues currently under debate regarding AR temporal characteristics.

In an effort to explore the behavior of the acoustic reflex in industrial environments, this investigator evaluated

changes in AR parameters following a two-hour industrial noise exposure. This evaluation consisted of several measures of AR properties including temporal characteristics, both onset and offset, AR threshold, magnitude, and adaptation. Potential relationships between various AR properties and expressions of temporary threshold shift were also investigated.

Review of the Literature

Temporary Threshold Shift

Various responses to noise have been reported in the literature (Davis, 1958; Dougherty, 1970; Kryter, 1970). The primary effect of noise on health, however, remains the production of noise-induced hearing loss. This connection between loud sound and hearing loss has been recognized for hundreds of years. The first reference noting the effect of noise on human hearing appears to be credited to Pliny the Elder, in about 600 A.D. (Ward, 1980a). He noted that persons living near the waterfalls of the Nile were "strucken deaf" from the loud roar of the water. Noise soon became one of the undesirable by-products of man's progress. Increasing noise exposure in our highly industrialized society has made the problem of noise-induced

hearing loss difficult to ignore. The effect of noise on the auditory system is now the subject of great concern and involves the efforts of many agencies and individuals.

Noise can result in permanent or temporary changes in hearing sensitivity. The principal method of investigating noise induced hearing loss is the measurement of threshold shift. Threshold shift is documented by measuring changes in hearing sensitivity prior to and following a specified noise exposure. Temporary threshold shift is known to recover within some time interval to pre-exposure levels. Permanent threshold shift on the other hand, is irreversible, and does not recover throughout the lifetime of the organism. As a rule, many of the same factors which influence temporary threshold shift (TTS) also affect the development of permanent threshold shift (PTS). Such factors as intensity, duration, and spectrum of the noise along with individual variability have been correlated to the development of both TTS and PTS. This is not to suggest that the underlying properties of TTS and PTS are identical, however, certain parallels do exist between the two. The inherent problem in studying PTS is the ethical responsibility of the researcher not to induce a permanent change in the auditory sensitivity of his subjects. The investigation of PTS, therefore, must be limited to animal models which only serve as approximations of the human auditory system. Although previous studies indicate that TTS cannot be used

to predict PTS (Mills, 1984; Ward, 1980a, 1980b), both are dependent on certain cochlear events which are predicated by parameters of the fatiguing stimulus.

The time interval between the end of the exposure and when threshold is measured represents a critical variable in the quantification of TTS (Melnick, 1978). The pattern of TTS recovery is complex and is influenced by the process of adaptation and sensitization of the auditory system. Hirsh and Ward (1952) first reported a polyphasic pattern of TTS recovery which occurs within the first few minutes following the cessation of the stimulus. They termed this rapidly changing recovery pattern the "bounce effect." As illustrated in Figure 1, there is a rapid recovery of threshold in the first minute following the cessation of the noise. This is then followed by an increase in threshold shift where it reaches a maximum level at approximately two minutes into the post-exposure period. Recovery then continues on a monotonic pattern until hearing threshold returns to pre-exposure levels. To avoid the complicated interaction of these changing auditory processes, TTS is usually measured at about two minutes post-exposure time (TTS_2).

The degree of temporary threshold shift (TTS) depends on many factors including the spectrum, intensity, duration, and temporal pattern of the fatiguing stimulus (Gerhardt, Melnick & Ferraro, 1979). Broadband stimuli typically

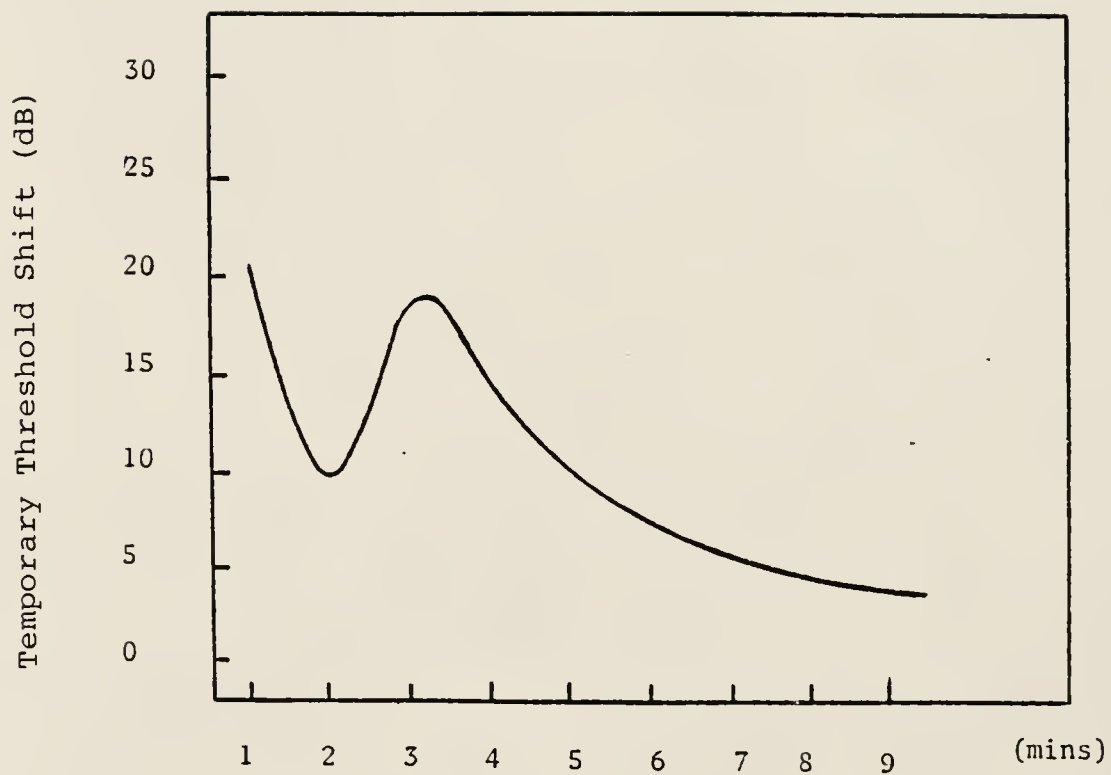


Figure 1. Polyphasic recovery pattern of temporary threshold shift (adapted from Hirsh & Ward, 1952).

have their greatest effect in the 3.0 to 6.0 kHz region. This is most likely due to several factors including resonance characteristics of the outer and middle ear, excessive mechanical stress on certain areas of the cochlear partition, and circulatory patterns within the inner ear itself (Wiener & Ross, 1946; Shaw, 1974; Tonndorf, 1976; Pickles, 1982). When discrete frequencies are utilized as the fatiguing stimulus maximum TTS is typically measured one-half to one octave above the exposure frequency (Davis, Morgan, Hawkins, Galambos, & Smith, 1950; Hirsh & Bilger, 1955). Ward (1973) has also reported that in general, high frequency noise exposures produce greater TTS than comparable exposure levels of lower frequency stimuli.

Another important factor influencing the development of TTS is the interaction between sound intensity and exposure duration. Melnick (1976) reports that TTS increases as intensity and duration of the exposure increase. When evaluating the effects of noise neither element can be considered alone. In an effort to describe the effects of exposure intensity level, however, we must assume that other variables, such as duration and frequency spectra, are held constant. Under these constraints TTS usually increases linearly with increments of sound pressure level (Melnick, 1978). This linear relationship exists only after the exposure exceeds a lower limiting intensity level of about 70 to 75 dB SPL (Ward, Cushing, & Burns, 1976). It

is also known that moderate intensity exposures create TTS that is proportional to the sound pressure level of the noise, while extremely high intensity stimulation may actually decrease the TTS developed (Davis, Morgan, Hawkins, Galambos & Smith, 1950; Miller, 1958). Ward (1968) speculates that this response may be due to a change in the way the stapes vibrates at the oval window after maximum contraction of the middle ear muscles.

Exposure duration represents another critical variable in the development of TTS. Experiments involving both human and animal subjects with exposures greater than eight hours have shown TTS to increase as duration of the exposure increases up to a certain level and then to plateau (Mills, Gengel, Watson & Miller, 1970; Melnick & Maves, 1974). Carder and Miller (1972) observed this TTS growth pattern in their study of chinchillas and coined the term "asymptotic" threshold shift (ATS). ATS has been reported to occur somewhere between eight and sixteen hours after exposure in human subjects. Idealized curves of TTS development for the maximally affected frequency as a function of exposure duration and intensity are illustrated in Figure 2 (Melnick, 1978). Note there is a lower intensity level, equivalent quiet, which does not produce TTS regardless of exposure duration. However, when this level is exceeded, TTS will grow linearly with logarithmic exposure duration up to eight to twelve hours and then level off at asymptotic

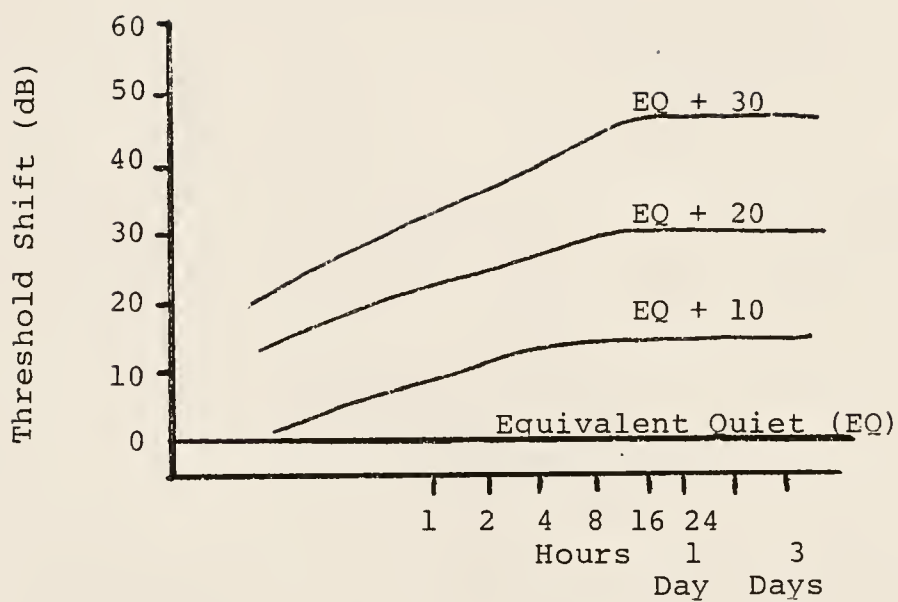


Figure 2. Idealized pattern of TTS as a function of exposure duration and intensity (adapted from Melnick, 1978).

threshold shift. It should also be apparent that the growth rate of TTS depends on the intensity of the noise as previously mentioned.

The time pattern of the exposure is another parameter which is known to influence the development of TTS. When the exposure consists of noise that is fluctuating or intermittent, a complicated relationship begins to develop between total duration of the noise exposure and the degree of TTS it produces. Under such circumstances the on-time and off-time of the fatiguing stimulus must be considered when relating the exposure to TTS. It can be assumed, from limited research, that intermittent sounds, in general, produce less TTS than continuous exposures of the same total duration. For particular exposure conditions, TTS has been found to be proportional to the ratio of time occupied by the sound to the total time of exposure. This relationship is known as the "on fraction rule" (Ward, 1970; Ward, Glorig & Sklar, 1958). For example, the on-fraction rule would predict that when a sound occupies only one-half of the total exposure time, the magnitude of TTS developed would be about one-half of that which would have been produced if the fatiguing stimulus had been continuous. It should be noted, however, that this relationship is applicable only for sound bursts of 250 msec to two minutes, and noise with frequency spectra above 1200 Hz (Selters & Ward, 1962).

Although susceptibility to TTS has been found to be normally distributed, the variance among individuals is quite large. Not only do differences exist between individuals for a particular noise exposure, but differences exist in the same person for different types of noise exposures (Ward, 1968). Standard deviations between subjects are typically in the range of 5-10 dB, while intrasubject variability reveal standard deviations of approximately 2 to 4 dB. Intersubject variation also becomes greater with increases in test frequency (Melnick, 1976).

Physiology of the Acoustic Reflex

The acoustic reflex is a bilateral non-voluntary response that occurs in the presence of intense acoustic stimulation. Although research indicates that the AR can be elicited by non-acoustic stimuli (Dallos, 1961), this section will deal with the auditory response of the acoustic reflex arc. Contraction of the middle ear muscles results in a change of impedance at the tympanic membrane and helps control the transmission of sound through the middle ear system. This control is thought to occur from an increase in stiffness and a functional decoupling of the middle ear structures.

Two middle ear muscles are involved in acoustic reflex action. Both the tensor tympani and stapedius are pennate muscles which, by design, are able to exert a great deal of tension with a minimal amount of displacement. The tensor tympani muscle is approximately 25 mm in length and rises from the cartilaginous portion of the eustachian tube. The muscle itself is enclosed in a bony capsule from which a tendon emerges. The tendon exits the bony enclosure and turns around the cochleariform process before inserting on the manubrium of the malleus. This muscle is innervated by a motor branch of the trigeminal nerve (CN. V) via the otic ganglion (McPherson & Thompson, 1977). Upon contraction the muscle pulls the malleus and tympani membrane inward (Jepsen, 1963).

In man, the stapedius muscle is chiefly responsible for the impedance changes observed at the tympanic membrane. The stapedius muscle is approximately 63 mm in length and arises within the pyramidal eminence. It is completely enclosed throughout its entire length in a bony capsule. A tendon from the muscle emerges from the pyramid, and inserts at the neck of the stapes. The stapedius muscle is innervated by a motor branch of the facial nerve (CN. VII) (McPherson & Thompson, 1977). Upon contraction the muscle pulls the stapes down and outward. This movement causes a stiffening of the ossicular chain which increases the impedance of the middle ear system (Jepsen, 1963).

The acoustic reflex arc is made up of neurons connected to muscle fiber. Borg (1973) described the reflex arc as being a very secure, short latency pathway. In addition to the direct neural tracts, there are probably numerous indirect, parallel, multisynaptic pathways. These neural pathways are largely unknown but may involve certain elements of the extrapyramidal system. The direct neuronal pathway consists of three to four neurons: the primary afferent neurons carry impulses from the hair cells to the cochlear nucleus; the second synapse is located at the ventral cochlear nucleus; the third neuron is in the superior olivary complex (SOC). The SOC has connections with both ipsilateral and contralateral nuclei of the facial nerve (CN. VII), which innervates the stapedius muscles (Borg, 1973).

The reflex arc contains motoneurons of different sizes, and diameters which dictate their particular conduction properties. Basic neuronal physiology indicates that in general, smaller diameter neurons will have slower conduction velocities when compared to larger diameter neurons. This premise appears to hold true for the reflex arc. The thinner motoneurons of the stapedius muscle are known to have lower thresholds and slower conduction velocities. They also, as a rule, connect to slower contracting muscle fibers. As a result of this anatomical arrangement, a weak input to the reflex system will excite

mainly small, slow conducting neurons which activate slow contracting muscle fibers (Borg, 1976). The intensity dependent rise-time of the AR is thought to result from an orderly recruitment of faster units in response to higher intensity input. Higher intensity stimuli excite larger diameter, fast conducting neurons, which in turn connect to fast contracting muscle fibers. Research indicates that as the stimulus intensity increases, rise-time of the response decreases. This finding is compatible with an orderly recruitment of neural elements, though other possibilities exist (Borg, 1976).

Borg (1972) has also been able to demonstrate that changes in middle ear impedance closely relate to EMG activity of the stapedius muscle. Good correlation was also demonstrated between the amplitude of the integrated EMG and magnitude of the AR response. In a related study Zakrisson, Borg, and Blom (1974) made simultaneous EMG recordings of the stapedius muscle in one ear while monitoring changes of acoustic impedance activity in the other ear. Results showed that at low intensity levels EMG activity directly related to changes of impedance at the tympanic membrane. Cessation of the eliciting stimulus, however, revealed an apparent "after-discharge" firing in the EMG activity for up to 400 msec. This finding may partially explain why offset latencies of the acoustic reflex are generally longer than onset responses.

The latency from stimulus onset to the first motor-unit action potential generally decreases with increments of stimulus intensity. As previously mentioned this may be the result of an orderly recruitment of motorunits with differing thresholds and conduction velocities. However, offset properties have been demonstrated to be relatively independent of stimulus parameters, and are generally longer in latency. As demonstrated by Zakrisson, Borg and Blom (1974) this may be due to basic physiology of the motor unit and after-discharge firings observed during EMG recordings. Therefore, from the evidence available, it appears that acoustic reflex latency characteristics can be explained by neural conduction properties and after-discharge firings of the individual motorunits. The results of this and other research seem to indicate that the acoustic reflex arc has properties commonly seen in other polysynaptic pathways (Borg, 1976).

Theories on Middle Ear Muscle Function

The middle ear of man is a sophisticated biological system that serves as an impedance-matching device between the sound-conducting medium of air and inner ear fluids. The structures involved in this transforming process are the tympanic membrane, ossicular chain, and middle ear

muscles. Understanding the function of the middle ear muscles has been the focus of scientific investigation for many centuries. Observations completed on both animal and human models have resulted in several hypotheses regarding the contribution of the intra-aural muscles to the process of audition. Borg, Counter, and Rosler (1984) have summarized the findings of numerous experiments into four major theories: 1) the intensity-control protection theory, 2) the ossicular chain fixation theory, 3) the accomodation-frequency selection theory, and 4) the labyrinthine pressure regulation theory.

1) Intensity control-protection theory

In the seventeenth century, Fabricius ab Aquapendente (1600) speculated that the function of the middle ear muscles was to prevent the tympanic membrane from rupturing during intense exposure to sound. von Helmholtz (1868) believed that the middle ear muscles caused a diminution in the sound pressure level transmitted to the inner ear labyrinth. Kato (1913) was one of the first to observe contraction of the middle ear muscles to moderate sound pressure levels. Because of his systematic approach to the study of the intra-aural muscles, the intensity-control theory emerged as the dominant theory of acoustic reflex physiology during the early decades of the century.

2) The ossicular chain fixation theory

This theory proposed that the major function of the middle ear muscles was to maintain ossicular chain position and continuity. The general view was that the muscles served no functional role in hearing, but were merely present to maintain the ossicles in their respective positions in a state of readiness for transmission of sound to the inner ear labyrinth (Borg, Counter, & Rosler, 1984).

3) The accommodation-frequency selection theory

The accommodation-frequency selection theory states that middle ear muscle contraction acts like a filter and allows selective transmission of certain frequencies through the middle ear system. Certain investigators believed that the tension exerted on the tympanic membrane by muscle contraction involuntarily tuned subject's ears to various sounds (Du Verney, 1683). Lucae (1874) assigned even more specific roles to middle ear musculature. He postulated that lower frequency sounds (below 6.0 kHz) were accommodated by tensor tympani contraction, and assigned the role of high frequency accommodation to the stapedius muscle. The most intriguing version of this theory was presented by Striker (1880), who theorized that the mere thought of sound or melody could cause involuntary activation of the middle ear muscles.

4) The labyrinthine pressure regulation theory

This theory states that contraction of middle ear muscles (mainly the tensor tympani), causes an increase in the pressure of labyrinthine fluids, and thereby dampens the acoustic energy reaching the inner ear. Kato (1913) discredited this theory after his series of systematic observations of the middle ear system. He found no pressure increase at the labyrinth during experimentally induced tensor tympani contraction.

While the intensity-control hypothesis is generally the theory of choice, certain arguments have been raised regarding the protective function of the middle ear muscles. One major objection is that the latency of muscle contraction is relatively slow and, therefore, cannot effectively attenuate many types of acoustic stimuli. Secondly, it is widely recognized that AR contraction fatigues quite rapidly to continuous intense sounds. Research also appears to suggest that only low frequency sounds are attenuated by middle ear muscle contraction, whereas noise-induced hearing loss occurs in the higher frequencies (3.0 to 6.0 kHz). For these reasons, the protective function of the acoustic reflex has been challenged. The protective theory has also been criticized from an evolutionary point of view. Many individuals argue that there are no natural environmental

reasons for the development of a system which protects the ear from intense twentieth century noise.

Although it is true that acoustic reflex is too slow for a protective function from single impulse sounds, this may not be the case for other types of noise exposures. It should be noted that rapid adaptation of the AR activity has typically been demonstrated for continuous intense stimuli (Djupesland, Flottorp & Winther, 1966). However, industrial exposures typically include a rapid succession of fluctuating or impulse type sounds. Under these circumstances the relaxation properties of the AR may be more important. Borg (1976) demonstrated the AR has the ability to reactivate and recover during industrial noise exposures thereby enhancing and preserving its attenuation properties. In an experiment utilizing shipyard noise, Borg, Nilsson, and Liden (1979) demonstrated only minor AR fatigue in workers following a full day of intense industrial noise exposure. In another study involving Bell's palsy patients, Zakrisson, Borg, Liden, and Nilsson (1980) revealed significantly greater TTS in the affected or paralyzed ear than in the ear with normal acoustic reflex activity. Greater hearing loss extending in the low and mid frequencies was evident in the ear without AR function. We can, therefore, speculate that auditory trauma would be even greater under certain conditions following noise exposure without the presence of the acoustic reflex system.

The evolutionary need of the AR may be demonstrated if one closely examines the intense vocalizations made by animals and man. The AR is known to be active during many self-stimulating activities such as talking, chewing, eating and screaming. Measurements made at 20 cm from the head of adults during voluntary loud vocalizations have been reported to reach 126 dB SPL (Borg, Counter & Rosler, 1984). These one-second vocalizations correspond to approximately 132 dB at 10 cm from the mouth, and have the same energy as a 15-minute exposure of noise at 102 dB Leq (Borg, Counter & Rosler, 1984). Therefore, when activated by self-stimulation or external stimuli, the middle ear muscles can help maintain the sensitivity of our auditory system. Without the action of the AR, auditory fatigue may occur even more rapidly, resulting in deterioration of auditory performance. Therefore, it seems reasonable to assume that the AR acts to increase the dynamic range of the auditory system and helps to maintain sensitivity in a variety of listening environments.

The AR may also serve to enhance speech perception abilities. As previously mentioned the chief effect of the AR activation is the attenuation of sound below 2.0 kHz. This frequency selectivity may provide a partial remedy for the masking effect low frequency vowel sounds have on higher frequency consonants. Research indicates that this low frequency attenuation may serve to improve speech perception

in many competing listening situations, and thereby improve communication ability (Zakrisson, 1974).

In light of the evidence presented, Borg, Counter and Rosler (1984) speculate that the function of the middle ear muscles may be quite extensive in many listening situations. They postulate that, under certain circumstances, the acoustic reflex can prevent interference, minimize injury, and improve auditory communication ability. If this is the case, the middle ear muscles appear to provide a unique mechanism to control auditory input which allows the organism to separate relevant from irrelevant sounds.

Acoustic Immittance

Acoustic immittance is the general term used to describe the transfer of acoustic energy through the middle ear system. This energy transfer can be expressed in terms of acoustic impedance or acoustic admittance (Wiley & Block, 1979). One of the most attractive aspects of acoustic immittance measurements is that clinical use of the equipment requires very little understanding of impedance or admittance concepts. The vast number of terms and procedural differences which exist from one manufacturer to the next causes a considerable amount of confusion to the student of impedance audiometry (Popelka,

1984). Audiologists should understand, at least on a conceptual basis, the physical principles involved in these measurements to minimize possible errors produced in their clinical and research efforts. Knowledge of the limitations of these measurements must also be considered in order to make full use of immittance technology.

The term impedance is used for mechanical, electrical and acoustic systems. Mechanical impedance is the ratio between an applied force and velocity with which the system is moved. Electrical impedance is the ratio of the applied voltage to the current flow. Acoustic impedance relates the applied sound pressure level to volume velocity. Volume velocity can be understood by considering a sound pressure being applied to a unit area of the eardrum. If one assumes that all the particles on that unit area, and in the air for a certain depth behind the eardrum, move in a uniform manner, then a unit volume will be set into motion with a certain velocity, the volume velocity (Bennett, 1984). Acoustic impedance then is the ratio between the sound pressure level present, and the volume velocity which is produced. Therefore, conceptually, acoustic impedance is a way of noting the physical response of a system. The unit of measure of acoustic impedance is the ohm. The acoustic ohm has been arbitrarily defined as occurring when a sound pressure of one dyne produces a volume velocity of air of one cubic centimeter per second (ANSI, 1960). In other words, an ohm

is a unit of measure that expresses the resistance to energy flow at the surface of the eardrum (Feldman, 1976).

Three inherent characteristics of impedance interact in a complex manner to determine the mobility of the middle ear system: mass, resistance and stiffness. Mass is related to the density of the elements of the system. In the middle ear, mass is composed of the weight of the ossicles, and the tympanic membrane. Resistance occurs whenever there is a change in the applied energy of a moving system to another form, usually heat. The resistance factor in the middle ear is minimal, due to the suspension of the ossicles by muscles and ligaments. Stiffness is the tendency of a system to retain its original shape and position. In the middle ear system, it is generally attributed to the motion of the stapes and resistance of the inner ear fluids. The effect of mass, friction, and stiffness generally determines the total impedance of the system (Northern & Grimes, 1978). Acoustic reactance is the imaginary component of impedance resulting from the stiffness and mass of the system, and is the component that expresses the storage and return of acoustic energy (Feldman & Wilber, 1976). The formula for impedance is presented below:

$$Z = \sqrt{R^2 + (2\pi f \cdot M - S/2\pi f)^2}$$

where Z = impedance, R = resistance, M = mass, S = stiffness, f = frequency, and $\pi = 3.14$ (Northern & Grimes, 1978).

Any mathematical description of impedance must also include the parameter of time. It is convenient to express time in angular fractions of one wavelength of applied energy. In Figure 3, we can see that a unit volume being displaced uniformly for a sinusoid signal actually has three separate characteristics of motion: displacement, velocity, and acceleration. The phase relationships of (a) displacement, (b) velocity, and (c) acceleration differ considerably over time. At the positive and negative peak of displacement (a) the motion stops momentarily before starting in the opposite direction, resulting in zero volume velocity at that point in time. The maximum velocity (b) occurs as the displacement passes through the baseline. The acceleration of the system (c) will be at its maximum when the rate of change in velocity is greatest (as the velocity curve passes through baseline). Examination of the curves show that displacement lags the velocity by 90 degrees, and acceleration leads it by 90 degrees. The relationships between these components of motion can now serve as references for impedance quantities.

If velocity is arbitrarily placed at 0 degrees phase angle, we can now discuss certain relationships of the middle ear system as they relate to the properties of motion. Recall, that the resistance of the middle ear

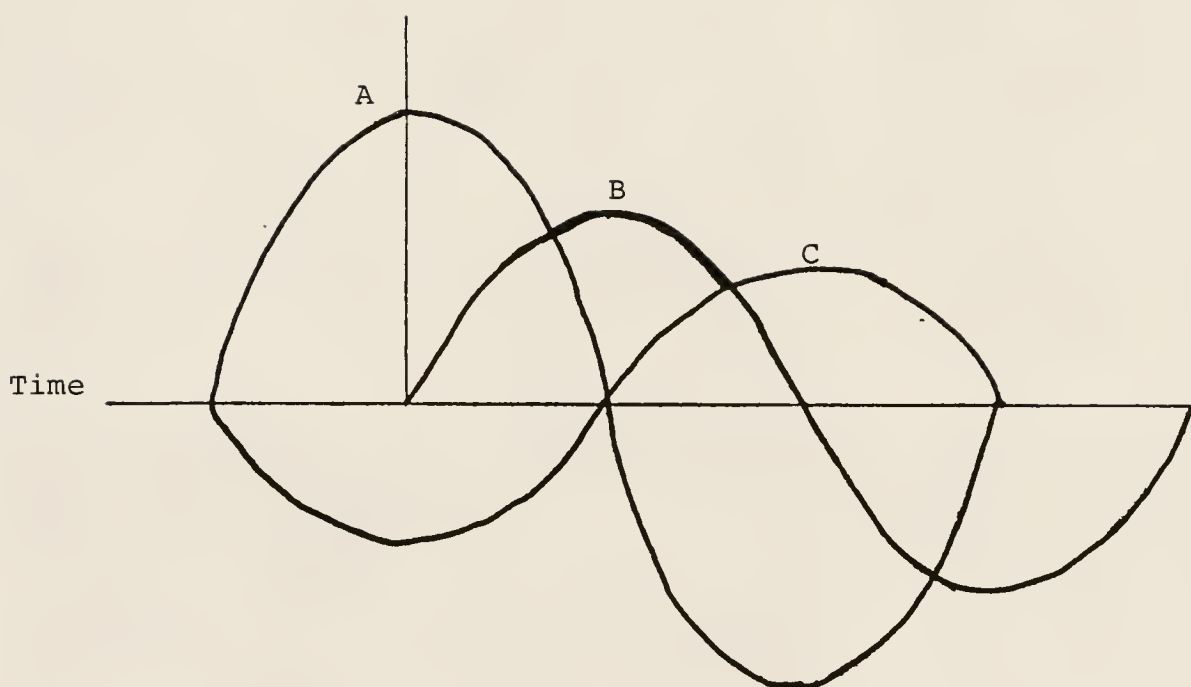


Figure 3. Sinusoidal curves for (a) displacement, (b) velocity, and (c) acceleration. Note that displacement lags velocity by 90 and acceleration leads the velocity by 90 (adapted from Bennett, 1984).

system is provided by the fluids in the cochlea, and is defined as an opposition to the flow of energy. This is similar to the opposition a spring offers to mechanical motion. Lagging the resistance by 90 degrees is stiffness reactance, X_s . This is analogous to displacement of a spring. The other reactive component, mass reactance (X_m), derives its properties from the mass multiplied by acceleration, and is the force required to overcome the inertia of the system. Figure 3 illustrates that the mass reactance, represented by (c) acceleration, is 180 degrees out of phase with the stiffness reactance X_s , represented by displacement (a). To compute the overall impedance of the system, the net reactance must simply be linked to the resistive component.

Vectors lend themselves to graphic representation and are mathematical descriptors of physical events which involve both magnitude and phase. Figure 4 demonstrates that the resulting vector has a length $|Z|$, which is the magnitude of the impedance without consideration to phase angle (0). Note if the net reactance ($X_m - X_s$) is very large when compared to the resistive component R , then the phase angle will be large. This is typically the case for a low frequency probe tone (220 Hz) where stiffness dominates, resulting in a phase angle towards -90 degrees (Bennett, 1984). It should be noted, however, that mass reactance X_m , is frequency dependent, and therefore, is affected by

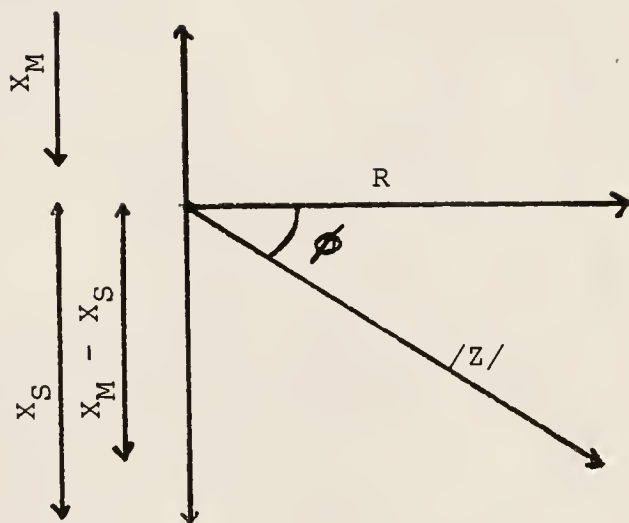


Figure 4. The vectoral components of impedance (adapted from Bennett, 1984).

probe tone frequency. Differing probe tones may have a profound effect on impedance vectors quantities. Also as frequency increases, the middle ear structures must move faster resulting in less displacement, therefore, the stiffness reactance (X_S) is reduced. All of these variables must be considered for any mathematical transformation involving impedance or admittance values (Block & Wiley, 1979).

While acoustic impedance provides a measure of a systems opposition to the flow of energy, admittance, which is the reciprocal of impedance, represents the ease with which sound is transmitted. Like impedance, acoustic admittance is a vector quantity and has two subcomponents, susceptance (B) and conductance (G). Acoustic conductance (G) represents the flow of energy through a resistance. Acoustic susceptance (B) is an expression of the storage of energy, and represents the reciprocal feature of reactance. The relationship between admittance (Y) and impedance (Z) is presented in Table 1 adapted from Northern and Grimes (1978).

Because conductance (G) is in phase with the pressure (displacement) rather than velocity, the positive susceptance (B) component is related to stiffness, and the negative one to mass. This results in positive vector relationships as shown in Figure 5. It should be noted, however, that conductance and susceptance are not merely inverses of resistance and reactance. Measuring both (G) and (B) has

Table 1
Immittance terminology
(adapted from Northern & Grimes, 1978)

Units of Measure	<u>Impedance</u>	Characteristics	<u>Admittance</u>	Units of Measure
	Terms		Terms	
Ohms	<u>Resistance</u>	Friction	<u>Conductance</u>	Mhos
	(R)		(G)	
	<u>Reactance</u>	Mass	<u>Susceptance</u>	
	Positive (Xa)		Negative (-Ba)	
	Negative (-Xa)		Positive (Ba)	

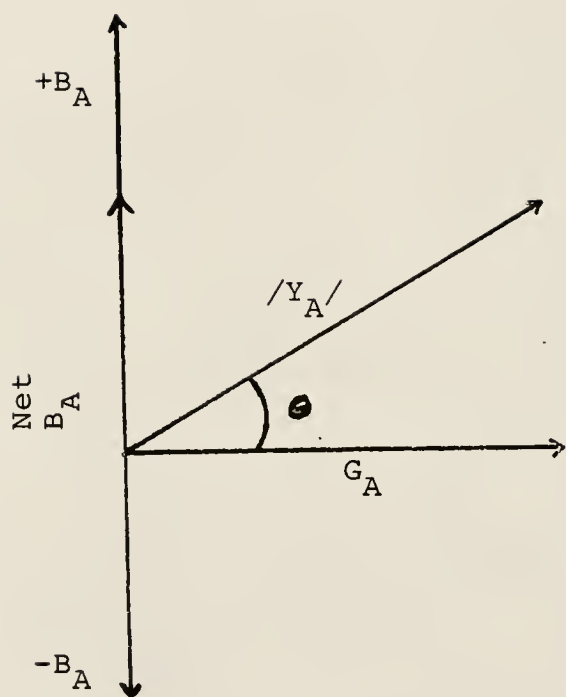


Figure 5. The vectorial components of admittance (adapted from Bennett, 1984).

distinct advantages when one is adjusting for ear-canal contribution to overall measurements made at the probe tip (Block & Wiley, 1979; Bennett, 1984). Therefore, after adjusting for ear-canal volume, and considering phase relationships, the overall admittance of the middle ear system can be determined with the following formula.

$$|Y| = \sqrt{G^2 + B^2}$$

Tympanometry is the measurement of compliance change as air pressure is varied in the external canal (Northern & Grimes, 1978). This procedure is extremely useful in determining eustachian tube function, and middle ear pressure values. In essence, the tympanogram is a measure of the relative change in sound pressure level within the ear-canal cavity. When the probe tone is introduced into the ear-canal sound waves hit the tympanic membrane. Some energy is reflected back at differing phase and amplitude. The difference in phase and amplitude between the probe frequency, and the reflected wave depends on the impedance (or admittance) characteristics of the middle ear system (Northern & Grimes, 1978).

Many clinicians conceptualize the tympanogram as the way the eardrum moves. In reality, the tympanogram is simply a recording of changes in sound pressure level in the external ear cavity between the probe tip and tympanic membrane. The point of maximum compliance of the system is

simply the point with the lowest sound pressure level as the clinician varies the pressure. The measurement of tympanometry is extremely important to the study of acoustic reflex behavior. Research indicates that conductive middle ear pathology, and abnormal middle ear pressure can serve to elevate AR thresholds, or even prevent their recording (Jerger, 1970; Zwislocki & Feldman, 1970; Jerger, Anthony, Jerger, & Maudlin, 1974).

Acoustic Reflex Threshold

Acoustic reflex threshold may be defined as the lowest stimulus level that produces a measurable change in acoustic immittance. It should be recognized, however, that the acoustic reflex threshold is influenced by many factors which have little to do with auditory performance or sensitivity. Such variables as the nature of the elicitor, equipment sensitivity, and manner with which AR activity is monitored all affect the final definition of acoustic reflex threshold.

Most investigators define AR threshold as the smallest admittance change from baseline activity that occurs in association with elicitor presentation (Gelfand, Silman, & Silverman, 1981; Popelka, Margolis, & Wiley, 1976; Peterson & Liden, 1972). Several authors have proposed a more

operational definition of AR threshold (Borg, 1972; Block & Wiley, 1979; Hepler, 1984; Moul, 1985). Borg (1972) defined AR threshold as the presentation level required to shift acoustic immittance from baseline to 10% of its maximum immittance change. Block and Wiley (1979) utilized a statistical technique for threshold determination. They choose to define AR threshold as the lowest elicitor level that causes an admittance change from baseline activity of more than two standard deviations. This technique has also been utilized with success by Moul (1985) and Hepler (1984).

The acoustic reflex is a bilateral response to intense stimuli which can be elicited by monaural or binaural stimulation. Ipsilateral or contralateral AR activity can be monitored in most laboratories; however, studies show that ipsilateral AR thresholds are approximately 5 to 8 dB lower than contralateral AR thresholds (Zakrisson, 1974). When the elicitor is presented bilaterally, there is approximately a 3 dB decrease in AR threshold (Moller, 1974). These variables must be taken into consideration when comparing results of one laboratory to another. The literature also reports high test reliability for AR threshold measurements (Chun & Raffin, 1979; Chermak, Dengerink & Dengerink, 1983). Forquer (1979) tested normal hearing and sensorineural loss subjects eight times over a period of three days and found largest AR threshold difference to be 2.4 dB.

At present, there are no apparent sex differences in AR threshold data for both normal and hearing impaired listeners (Jerger, Jerger & Mauldin, 1972; Osterhammel & Osterhammel, 1979; Silverman, Silman, & Miller, 1983). These authors also showed that AR thresholds are elicited at lower intensity levels with increasing age. It should be noted, however, that differences are only encountered if one compares extremely wide age groupings.

The acoustic reflex is also extremely dependent on the parameters of the eliciting signal. Stimulus bandwidth and density have been reported to be critical factors in AR threshold determination. Research indicates that AR threshold remains constant as stimulus bandwidth widens, up to a point. If the bandwidth of the elicitor is widened beyond that point, AR threshold is lowered (Moller, 1972; Djupesland & Zwislocki, 1973; Flottorp, Djupesland, & Winther, 1971; Margolis, Dubno & Wilson, 1980; Popelka, Margolis & Wiley, 1976). It is also worth noting the bandwidth for AR threshold increases as stimulus frequency increases (Flottorp, Djupesland, & Winther, 1971; Popelka, Margolis & Wiley, 1976).

Spectral density within a signal bandwidth also affects the AR threshold. Popelka, Margolis and Wiley (1976) were able to demonstrate this point by experimentally increasing and decreasing the number of components in a particular bandwidth signal. The larger the number of

components in the bandwidth of the signal, the greater its spectral density. Whenever the spectral density of the activating stimulus was increased a decrement in AR threshold was observed. It therefore appears, the critical band concept in acoustic reflex function is quite similar to other types of auditory performance.

A relationship also exists between the level, and the duration needed to elicit the acoustic reflex. The eliciting stimulus must have a certain duration and intensity level in order to cause middle ear muscle contraction. Reduction of elicitor duration below approximately 300 ms must be offset by increases in sound pressure level in order to reach AR threshold. This phenomenon by which the auditory system appears to integrate energy within a certain time frame is known as temporal integration (Gelfand, 1984).

Johnsen and Terkildsen (1980) utilized a series of click stimuli to demonstrate the temporal integration properties of the acoustic reflex system. They reported that AR threshold for a 128/sec click train was approximately the same as AR threshold for white noise in a group of normal listeners. However, as the click rate was varied from 128/sec to 8/sec a change in AR threshold from 75 dB to 120 dB was observed. This study seems to suggest that the auditory system integrated the fast click train as a continuous signal. When the click rates were significantly reduced, it prevented the individual clicks from being

processed in this fashion. Similar findings have been reported for tonal stimuli (Barry & Resnick, 1976; Richards, 1975; Woodford, Henderson, Hamernik, & Feldman, 1975). These findings appear to indicate that temporal integration properties of the acoustic reflex system are similar to those found in other types of auditory behavior.

Acoustic Reflex Magnitude

Acoustic reflex magnitude can be defined as the amount of immittance change resulting from the contraction of the middle ear muscles (Silman, 1984). Dynamic properties of AR magnitude have typically been demonstrated utilizing acoustic reflex growth functions. These graphs are a plot of immittance change resulting from increases in stimulus intensity level. Investigations reveal that AR magnitude is directly related to stimulus intensity level (Wilson & McBride, 1978; Silman & Gelfand, 1981; Moller, 1962). Stimulus parameters such as elicitor frequency, mode of presentation, and probe tone frequency have also been shown to influence AR magnitude (Wilson & McBride, 1978; Moller, 1962; Silman, 1984). Another factor which greatly influences the magnitude of the AR is the static immittance at the tympanic membrane. In general, as the static immittance of the system increases, so does the measured AR magnitude (Wilson, 1979; Block & Wiley, 1979).

Various normalization techniques have been used to describe AR magnitude functions. Borg (1977) and Moller (1962), expressed AR magnitude as a percent relative to maximum impedance change. Silman and Gelfand (1981) transformed the amount of impedance change to static acoustic impedance in decibels. The reporting of growth functions also differs in the literature. Several investigators describe acoustic reflex growth functions in terms of dB SPL (Moller, 1962; Borg, 1977), while others choose to relate magnitude re: dB above AR threshold (Djupesland, 1967; Thompson, Sills, Recke, & Bui, 1980; Gerhardt & Hepler, 1983; Hepler, 1984; Moul, 1985). Since saturation of the acoustic reflex appears to be governed by SL re: AR threshold rather than SPL of the elicitor, the latter method seems to be the most appropriate reference of the two. Such variables must be considered when comparing results from one laboratory to the next.

AR magnitude is dependent on the nature of the eliciting signal. Moller (1962) examined the effect of bilateral stimulation as well as ipsilateral vs. contralateral stimulation on AR growth functions. Findings indicate that AR slope functions were steepest for bilateral stimulation, and shallowest for the contralateral condition. Probe tone frequency has also been shown to influence AR magnitude functions. Dallos (1964) along with Wilson and McBride (1978) reported that for a given intensity level, AR magnitude

decreases with increases of probe tone frequency. These same authors also reported that AR magnitude was greatest for broadband noise and 1.0 kHz elicitors.

AR magnitude also varies as a function of age. Thompson, Sills, Recke, and Bui (1980) reported that growth of AR magnitude decreases for both tonal and noise stimuli as age increases. Silman and Gelfand (1981) and Wilson (1981) also found a decrease in AR magnitude for older populations.

The dynamic range of the acoustic reflex varies with the spectral content of the elicitor. AR dynamic range is approximately 30 dB for tonal stimuli (Dallos, 1964; Silman, 1984) and 50 dB for broadband noise (Wilson & McBride, 1978). It should be noted, however, that in many instances equipment limitations and subject discomfort prevent complete analysis of acoustic reflex growth functions.

Temporal Characteristics of the Acoustic Reflex

Onset latency is the time (in msec) of the first stimulus related immittance change resulting from middle ear muscle contraction. Measurement of AR latency depends on many factors including, characteristics of the eliciting stimulus, instrument response time, and mode of presentation. Latency values are also influenced by the operational definitions utilized by researchers. Borg (1972) and Moller

(1972) define onset latency as the time between stimulus onset and the point where the AR reaches 10% of its maximum amplitude. Colletti (1975) utilizes 5% of maximum immittance change as his definition of onset latency. Other investigators suggest that any measurable immittance change from baseline activity could be used to define onset latency (Metz, 1951; Sunderland, 1974).

Several methods are currently available for the study of AR activity. The choice of method also has certain implications for AR latency measurements. Examples of recording techniques include electromyography (Perlman & Chase, 1939), cochlear microphonics (Gerhardt, Melnick & Ferraro, 1979) and optical detection (Gans, Sweetman, & Carlson, 1972). While measurements obtained using these methods may be very precise, they require access to the middle ear or round window, and are generally impractical for clinical testing. Immittance measurements by far represent the easiest, most popular, and least invasive method available for the study of AR activity. Unfortunately, the time constant of these instruments often produces a systematic delay resulting from the time needed by the circuitry to perform their particular measurements. All latency values obtained must represent actual biological AR latency times when reported. This can be accomplished by subtracting the time constants attributed to the instrument involved in the measurements, from total latency values

(Bostra, Russolo, & Silverman, 1984; Lilly, 1984). Unfortunately the time constants vary depending on make and model of the instrument, and may even among individual pieces of equipment. Procedures used to obtain instrument response times of the equipment utilized in this study are presented in the methods section.

Several stimulus parameters directly affect onset latency values. Acoustic reflex latency is reported to be inversely related to stimulus intensity level (Metz, 1951; Borg, 1972; Dallos, 1964). The more intense the eliciting stimulus, the shorter the onset latency of the response. Rise-time of the activator has also been correlated with changes in onset latency. Studies show that the faster the rise-time of the stimulus, the shorter the latency of the response (McPherson & Thompson, 1977). Duration of the activating elicitor does not seem to affect latency values until it is shortened below a critical value. Whenever the activating stimulus has a duration of less than one second, the intensity of the stimulus needed to elicit the AR must be increased, and therefore, indirectly affects AR latency values (Djupestrand & Zwislocki, 1971). The effects of frequency on latency characteristics are at present equivocal. According to Moller (1958) elicitor of 500, 1000 and 2000 Hz are the frequencies of choice for the study of AR latency properties. Bosatra, Russolo, and Silverman (1984), however, point out that the use of low

and mid frequency tonal elicitors may be best simply because of their increased stability over other types of eliciting signals.

Latency characteristics of the AR are often discussed in terms of sensation level (re: AR threshold). Onset latency values for normal hearing young adults (20 to 30 years old) have been reported to be in the range of 150-250 msec at threshold (Metz, 1951; Moller, 1958; Dallos, 1964). When stimulus intensity levels are raised to 30 or 40 dB above AR threshold, latencies decrease to approximately 25-40 msec (Moller, 1984; Dallos, 1964). As with other AR parameters, AR temporal characteristics are extremely variable across individuals. Intrasubject variability, however, appear to be quite good. Bostra, Rossolo, and Silverman (1984) measured AR latency in 20 subjects twice a day for three consecutive days and found mean intrasubject variability to be only 9.7 msec, with a maximum variability in the order of 40 ms. In 40% of the subjects, AR latency remained constant over the entire three day period.

The dependence of onset latency to stimulus parameters does not hold true for all AR properties. While onset responses are inversely proportional to the intensity of the stimulus, offset responses show little or no intensity dependence (Borg, 1976). McPherson and Thompson (1977) suggested this nonlinearity may be due to inherent differences in the process of contraction vs. relaxation of the

AR response, and therefore, should be considered as two separate events of AR activity. They went on to postulate that the AR was actually an "energy related phenomenon." Since stimulus rise-time and intensity level affect the total energy available to the system, it seems only logical these properties control the onset latency of the response. Offset latency on the other hand, is relatively independent of stimulus parameters, and is thought to represent the system's response to cessation of the stimulus. Borg (1976), therefore, suggests that AR offset properties represent a closer estimate of neural conduction time of the AR system and should be investigated further.

Acoustic Reflex Adaptation

Magnitude of the acoustic reflex does not remain constant over time. Research indicates that when the activating signal is presented even for several seconds the middle ear muscles begin to relax, and immittance values change toward the static values that existed prior to stimulus onset (Wilson, Shanks, & Lilly, 1984; Fowler & Wilson, 1984). Several problems are typically encountered during the recording of acoustic reflex adaptation, which will be defined as the decrease in AR magnitude over time for sustained and interrupted signals.

The combination of procedural differences, instrument sensitivity, and baseline drift all contribute to the variability reported in the literature.

Adaptation is often quantified as the time (in msec) until the reflex decreases to 50% of its maximum reflex amplitude (Anderson, Barr & Wedenberg, 1970), or as a percentage of maximum amplitude at specified time intervals (Wilson, Shanks, & Lilly, 1984). Shanks (1979) reported that regardless of the protocol implemented, magnitude of the AR adaptation will depend on the unit of measure utilized to report the immittance change over time (admittance versus impedance). Antablin, Lilly, and Wilson (1980) demonstrated the effect of differing measuring units by plotting mean data functions expressed as percentage of maximum admittance and impedance. Results indicate that the impedance functions were slightly steeper than the admittance functions. This resulted in a shorter half-life values for the impedance (13.9 seconds) versus the admittance data (15.4 seconds). Such variation could have significant consequences under certain research conditions.

Baseline drift represents still another problem in the quantification of AR adaptation. Wilson, Steckler, Jones, and Margolis (1978) reported that acoustic admittance of the middle ear is constantly changing. During the measurement of reflex adaptation, middle ear pressure may decrease

slightly, resulting in a concomitant decrease in acoustic admittance at the tympanic membrane. Separating the contributions of instantaneous changes in middle ear pressure from the adaptation process represents a difficult obstacle in the measurement of AR adaptation. Tonndorf and Khanna (1968) suggest this baseline drift can be explained by an interaction of the middle ear mucosa and the eustachian tube. As the eustachian tube opens and closes, the middle ear pressure is equalized to ambient air pressure. The middle ear mucosa soon begins to absorb the air, creating slight negative pressure. The baseline drift demonstrated during adaptation measurement can, therefore, be explained as a series of tympanograms over time.

The rate of AR adaptation depends on the frequency of the activating signal (Djupesland, Flottorp, & Winther, 1967; Johansson, Kylin, & Langfy, 1967; Wilson, Shanks, & Lilly, 1984). In general, lower frequency signals (500 and 1000 Hz) produce less adaptation than higher frequency signals (3000 and 4000 Hz). Mid-frequency activators (1500 and 2000 Hz) exhibit varying degrees of reflex adaptation (Wilson, Shanks, & Lilly, 1984). Djupesland, Flottorp and Winther (1967) were one of the first investigators to demonstrate that AR adaptation was frequency dependent. They examined the duration of maintained AR activity for 250, 1000, and 4000 Hz elicitors. Results show that AR activity was maintained the longest for the 500 Hz

elicitor, followed by the 1000 Hz signal. The 4000 Hz elicitor had the fastest adaptation of approximately 20 seconds when presented at 10 dB SL (re: AR threshold).

Similar findings were reported by Johansson, Kylin, and Langfy (1967) utilizing 500 Hz and 3000 Hz elicitors. These authors reported little adaptation for the 500 Hz activator over a 10 second period, with substantial adaptation occurring for the 3000 Hz signal. Figure 6 from Wilson, Shanks, and Lilly (1984) demonstrates this frequency dependence with admittance data that has been corrected for ear canal volume (presented at 10 dB SL re: AR threshold). These data demonstrate that AR magnitude as well as adaptation properties are dependent on the frequency characteristics of the signal. As illustrated, the largest magnitude observed is for the mid-frequency signals. The 2000 Hz activator produced the largest magnitude and the 4000 Hz produced the smallest. Also note that virtually no AR adaptation is present for the low frequency elicitors (500 and 1000 Hz).

AR adaptation appears to be more resistant for noise activators than for tonal stimuli (Ward, 1961). This may be due to the random nature of noise stimuli which serves to re-elicite the AR response (Ward, 1973). In a study utilizing octave band noise elicitors, Djupesland, Flottorp, and Winther (1966) reported that low frequency noise was more effective at maintaining AR activity than higher

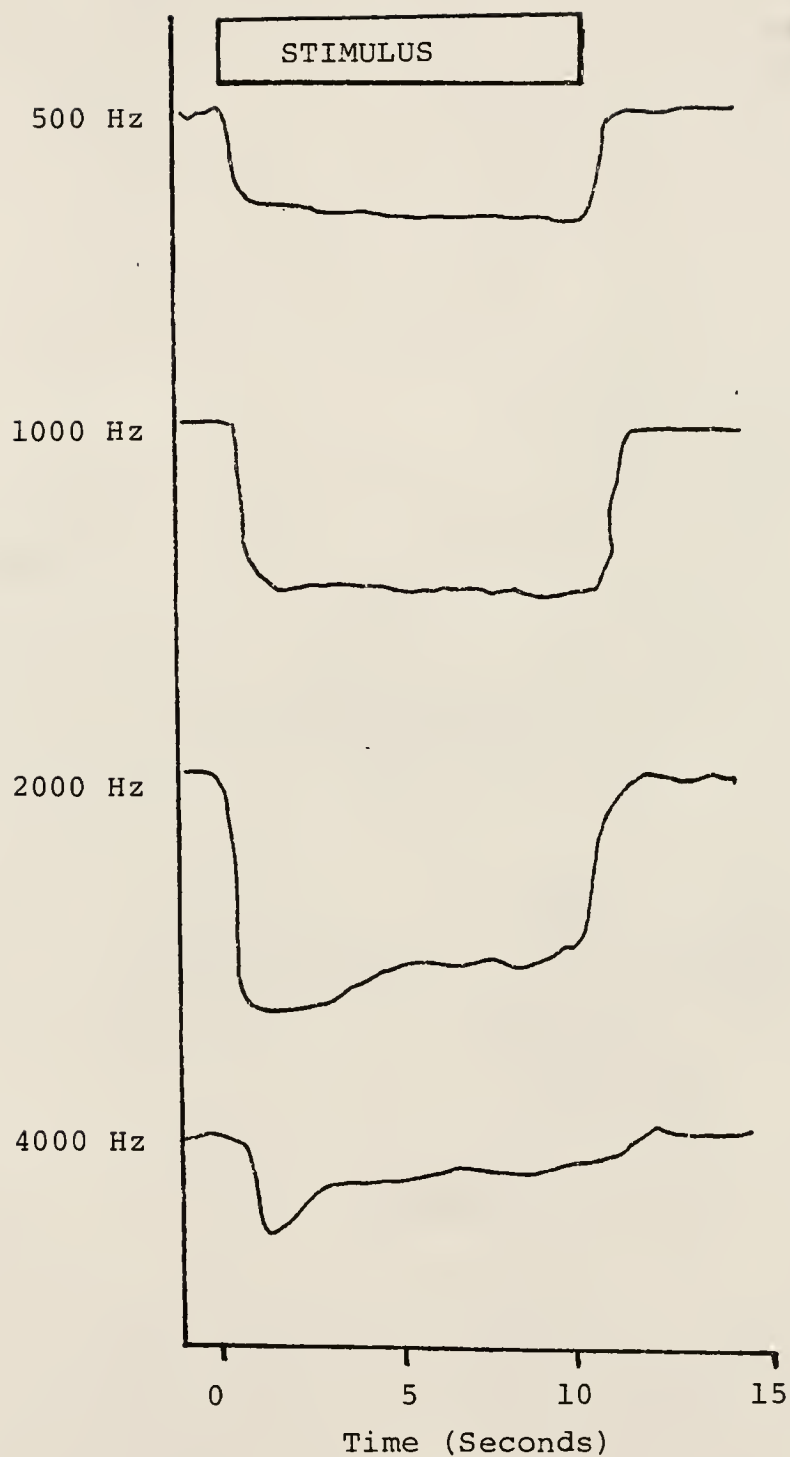


Figure 6. Adaptation functions obtained with four 10.2 s reflex-activator frequencies presented at 10 dB SL (adapted from Wilson, Shanks, & Lilly, 1984).

frequency octave-band noise. This finding may be related to the temporal encoding properties of the auditory system (Wilson, Shanks, & Lilly, 1984).

The relationship between AR adaptation and intensity level remains equivocal. Djupesland, Sunby, and Flottorp (1967) reported that higher intensity levels sustain AR activity for greater periods of time. Their results supported a direct relationship between intensity level and AR adaptation. Wiley and Karlovich (1975) on the other hand, reported that for a 500 Hz signal, reflex adaptation increased with subsequent increases in intensity level. Results to a 4000 Hz elicitor revealed no significant difference in AR adaptation as intensity level of the stimulus was varied. Wilson, Steckler, Jones and Margolis (1978) observed that there is a direct relationship between AR adaptation, and activator intensity level for 500 and 1000 Hz elicitors. This relationship became less pronounced, however, for higher frequency signals (3000 and 4000 Hz). In summary, most investigators report that AR adaptation decreases as the intensity level of a low frequency elicitor (500 and 1000 Hz) increases. Activators of higher frequencies (2000 Hz or above) reveal no systematic change in reflex adaptation properties with changes in activator intensity level (Wilson, Shanks, & Lilly, 1984).

Unfortunately much of the existing literature describing AR properties involves the use of constant pure tone or noise elicitors (Dallos, 1964; Johansson, Kylin, & Langfy, 1967; Anderson, Barr, & Wedenberg, 1969). These studies demonstrate rapid adaptation to a variety of acoustic stimuli. Research indicates, however, that AR activity can be reactivated with short pauses, or changes in spectral characteristics of the eliciting signal (Borg & Odman, 1979; Hetu & Careau, 1977; Gjaevenes & Sohoel, 1966). It is also known that typical industrial environments create noise of wide frequency content, and time varying intensity levels. In light of this fact, several investigators have evaluated AR dynamic properties in response to industrial noise.

Borg, Nilsson, and Liden (1979) exposed subjects monaurally to a 30 minute tape of shipyard noise while continuously monitoring AR activity. The first and last minute of the exposure were identical in order to evaluate the short term fatigability of the AR. Results indicate that AR properties were remarkably resistant to adaptation for this type of exposure. In a related study, these same authors evaluated AR performance following a typical workday of shipyard noise exposure. Employees were monaurally exposed at their worksite for approximately seven hours with periodic breaks. AR activity was monitored prior to, and immediately following the industrial exposure. Their findings revealed that the acoustic reflex shows very little adaptation following long term exposure to industrial noise.

The apparent discrepancy between studies involving industrial versus continuous noise exposures may be related to the temporal properties of the AR, and the transient nature of most industrial environments. While it is well established that single impulse sounds pass through the middle ear system unattenuated, they may affect the succeeding stimuli thereby providing some protective influence to the inner ear mechanism. As previously mentioned, the offset properties of the AR are much slower than the contraction process. Estimates based on impedance measurements indicate that offset times needed to reach 50% of maximum amplitude are in the order of 100-500 msec, and may exceed one second under certain conditions (Borg, 1976; Dallos, 1973). These findings along with the results obtained from such studies as Borg and Odman (1979) and Hetu and Careau (1977) suggest that when changes of intensity or frequency occur, as they do in most industrial situations, the AR undergoes a series of relaxation responses followed by reactivation. Due to this action, the AR may be able to maintain tension on the ossicle chain for longer periods of times without significant adaptation. Therefore, offset properties of the AR may represent a critical variable in the system's protective function against hazardous noise exposures. Further studies involving industrial noise exposures are needed, however, to validate these assumptions.

The mechanism of AR adaptation is presently unknown. Research indicates that neurons in the peripheral auditory system are characterized by a certain amount of adaptation. Kiang, Watanabe, Thomas, and Clark (1965) reported that neurons initially respond to a toneburst with an increase in firing rate that decreases with time. This process is evident throughout the auditory system. Recall, however, that low frequency activators demonstrate less adaptation than higher frequency stimuli (Wilson, Shanks, & Lilly, 1984). The fact that there is minimal adaptation when the AR is elicited by low frequency stimuli indicates that response amplitude may not be related to discharge rate, but rather, to other properties of neural activity (Moller, 1984). It has been suggested that the reflex response may be related to a phase-locking phenomenon, which have no adaptation properties (Kiang, 1980). However, additional research is needed to support this theory.

Acoustic Reflex Properties and Temporary Threshold Shift

Properties of the acoustic reflex, and their relationship to TTS, have previously been investigated in human and animal models (Gerhardt & Hepler, 1983; Borg, Nilsson, & Engstrom, 1983; Karlovich, Luterman, & Abbs, 1972; Gerhardt, Melnick, & Ferraro, 1979). It is well known that contraction of the stapedius reduces the transmission

of sound through the middle ear system. This reduction is greatest for the frequencies below 1 kHz and can be as much as 20 dB (Dallos, 1973). Due to this action, the acoustic reflex is thought to provide a protective function to the delicate structures of the cochlea. This reflexive response changes with various parameters of the stimulus including frequency spectrum, intensity, temporal pattern and duration (Gerhardt, Melnick, & Ferraro, 1979). However, the protective role of the acoustic reflex remains equivocal.

Johansson, Kylin and Langfy (1967) exposed normal hearing subjects to octave band noise, and found correlations between TTS developed, and certain latency characteristics of the AR. Holmes (1978), utilizing a white noise exposure, revealed significant correlations between AR magnitude at 2.0 kHz and TTS. Karlovich, Luterman and Abbs (1972) devised a unique experiment that incorporated a dichotic listening paradigm, to ensure AR contraction in an experimental versus control group of normal hearing subjects. The experimental group was exposed to noise in one ear, while an AR activating stimulus was presented to the other ear. Similar exposures were conducted for the control group without the AR activating tones present. The author reported significantly greater TTS in the control group when compared to the experimental group after the 1.0 kHz exposure. Zakrisson (1974) evaluated AR properties in

subjects with unilateral Bells palsy. This condition is known to cause paralysis of the stapedius muscle on the affected side. His findings indicated that TTS was significantly greater in the affected ear when compared to the non-affected ear following low frequency noise exposure.

Contrary to these findings Fletcher and King (1963) observed no significant difference in TTS developed in a group of stapedectomized patients when compared to the normal hearing control group. Turner (1974) also questioned the protective role of the AR. In his study he measured absolute impedance values and correlated them to TTS in a group of normal hearing subjects. Again, no relationship was observed between the impedance values measured, and threshold shift produced by the noise exposure. It should be noted, however, that these studies involved exposures of short duration (minutes) at high intensity levels. In an effort to obtain more a representative sample of AR activity, experiments utilizing exposures of greater duration have been completed.

Gerhardt, Melnick and Ferraro (1979) utilized an eight-hour, 95 dB SPL exposure of 0.5 kHz octave band noise on chinchillas to evaluate the relationship of the acoustic reflex to TTS. Round window recordings of the cochlear microphonic were used to study properties of the middle ear muscles. Measurements of reflex thresholds were obtained

prior to exposure, during quiet intervals of the exposure, and after cessation of the noise. Results showed that acoustic reflex thresholds were significantly altered by the noise exposure. Following eight-hours of exposure the average reflex threshold shift (RTS) was approximately 14 dB. The authors also concluded that the development of TTS and RTS followed the same time course. Specifically, for every 2 dB of TTS there was a 1 dB reflex threshold shift.

Gerhardt and Hepler (1983), in a study involving human subjects, measured middle-ear muscle activity with an electroacoustic bridge. This study investigated reflex threshold shift, and the subsequent development and recovery of TTS. The experimental group was exposed to a four-hour, 1.0 kHz noise in sound field at an intensity of 95 dB SPL. Results revealed that AR magnitude at suprathreshold levels decreased and recovered with the growth and recovery of TTS. This finding seems to suggest, that there is a relationship between AR magnitude and temporary threshold shift.

Hepler (1984) and Moul (1985) demonstrated that average AR magnitude is inversely related to certain expressions of TTS following two-hour exposures of broadband, and octave band noise, respectively. While certain AR parameters demonstrated to be significantly correlated to expressions of TTS, only a slight predictive relationship was demonstrated.

Statement of Purpose

Research indicates that certain parameters of AR function change following noise exposure, most notably, AR threshold and magnitude. Changes in onset latency have not been reported in the literature. One AR parameter which has not been studied extensively following noise exposure is the relaxation or offset response of the acoustic reflex system. There is also very little information regarding the adaptation process of the AR to industrial noise exposure. While continuous noise exposures may answer some of the questions regarding AR physiology, they can not explain the behavior of the acoustic reflex system in noise with changing temporal and spectral content.

As previously mentioned, single transient sounds pass through the middle ear system unattenuated. In industrial situations, however, impulses often occur as a series of fluctuations in background noise. Due to its relatively slow relaxation time, the AR offset response may be particularly important in these environments. If interimpulse intervals are brief, the contraction elicited by each impulse or intensity change, may affect succeeding stimuli. Under these conditions, offset properties may represent a critical variable in understanding the behavior of the acoustic reflex during industrial noise exposures. In an effort to explain some of the uncertainties concerning

AR behavior in industrial environments, the following questions have been formulated.

- 1) Do differences exist between pre-exposure and post-exposure values of the following response variables in normal hearing subjects after a two hour industrial noise exposure of 90 dB SPL?
 - a) Behavioral thresholds at octave and half-octave intervals
 - b) Acoustic reflex threshold
 - c) Acoustic reflex magnitude
 - d) Acoustic reflex latency characteristics (onset and offset)
 - e) Acoustic reflex adaptation
- 2) Do differences exist in percent adaptation for the three stimuli evaluated in the pre-exposure session?
 - a) Continuous broadband noise
 - b) Industrial noise
 - c) Interrupted broadband noise
- 3) Do any of these measures correlate to changes observed in audiometric behavioral thresholds following this same noise exposure?

CHAPTER II METHODS

This study evaluated changes in acoustic reflex activity following a two-hour exposure to industrial noise. Several parameters of AR activity were sampled prior to, and following the noise exposure in an effort to explain acoustic reflex behavior in industrial environments. As outlined in Table 2, the initial experimental session consisted of basic screening for subject selection, and the establishment of a baseline audiogram. During the second session, comprehensive analysis of the middle ear system and AR activity was completed. The third session included the industrial noise exposure, followed by measurement of acoustic reflex activity and post-exposure threshold testing. Recovery thresholds were also conducted 24 hours after the noise exposure. This was done in order to document complete recovery of behavioral thresholds.

Table 2
Experimental procedures.

Session One:

- 1) History and consent form
- 2) Otoscopic screening
- 3) Tympanometry
- 4) Acoustic reflex screening (BBN, thresholds < 85 dB SPL)
- 5) Subject Training--Beckesy tracking procedure
- 6) Audiometric Baseline--0.5 through 6.0 kHz

Session Two:

- 1) Otoscopic screening
- 2) Tympanometry
- 3) Acoustic reflex threshold (BBN stimuli)
- 4) Acoustic reflex magnitude/latency function (BBN stimuli)
- 5) Standard Adaptation (BBN stimuli) at 15 dB SL
- 6) Standard Adaptation (Industrial noise stimuli) at 15 dB SL
- 7) Reflex Interruption Test (BBN stimuli) at 15 dB SL

Session Three:

- 1) Pre-exposure threshold--0.5 through 6.0 kHz
- 2) Noise exposure (Industrial Noise--Two hours)
- 3) TTS at discrete frequencies 0.5-6.0 kHz (2 minutes following the exposure)
- 4) Acoustic reflex threshold (BBN stimuli)
- 5) Acoustic reflex magnitude/latency function (BBN stimuli)
- 6) Standard Adaptation (BBN stimuli) at 15 dB SL
- 7) Reflex Interruption Test (RIT) at 15 dB SL
- 8) Behavioral Threshold--0.5 through 6.0 kHz

Session Four:

- 1) Recovery Thresholds--0.5 through 6.0 kHz
 - 2) Recovery Acoustic Reflex magnitude/latency functions (BBN stimuli)
-

Subjects

Subjects were between 20 and 32 years of age, and were paid for their participation in this study. Thirty subjects were selected from students and university staff who met the following criteria: 1) normal hearing (≤ 15 dB HL re: ANSI, 1969) at octave and half-octave intervals from 0.5 through 6.0 kHz; 2) a normal tympanogram and otoscopic screening; 3) no history of chronic ear disease; 4) no employment history which required exposure to hazardous noise for periods in excess of two years; 5) AR thresholds of 85 dB SPL or less to a broadband noise elicitor; 6) ability to perform standard Bekesy procedure (modified method of adjustment) defined by repeatable thresholds (within ± 3 dB) at 1.0 kHz and stable tracings for up to one minute; 7) Bekesy excursion widths of less than 10 dB; 8) signed consent to the experimental procedures, plus verbal commitment to return in 24 hours following the noise exposure to assure complete recovery from TTS.

Instrumentation and Procedures

Exposure

The two hour exposure consisted of industrial noise presented bilaterally at 90 dB SPL. The noise sample was

obtained from a metal workshop in order to approximate an exposure commonly found in industrial environments. The noise consisted of background activity produced by several pieces of equipment, however, the main noise source was a piece of steel on a stone grinder. A 1/2" microphone (Bruel & Kjaer, type 4165), and sound level meter (Bruel & Kjaer, type 2203) was connected to a tape recorder (Teac, X-10/X7) which stored the noise sample. This signal was then delivered to an IBM-PC microcomputer with PCLAB real-time software package. This system facilitated the conversion of analog signals into retrievable data files. One second of industrial noise activity was sampled at a rate of 12.0 kHz to create the digitized file used for the noise exposure. The file was then stored to disk.

Upon keyboard command, the file was accessed, fed through a D/A converter, shaped with spectrum equalizer (Realistic 31-2008), amplified (Coulbourn Instruments, model S82-24 mixer-amplifier), attenuated (Hewlett-Packard, 350D attenuator set), and fed through Beyer DT49 earphones in an Industrial Acoustics audiometric booth. The subjects were in a reclining chair for the duration of the two hour noise exposure.

A fast-fourier transform (FFT) completed on stimuli used to produce the TTS is included in Figure 7. Also presented is a sample of broadband noise processed through the same instrumentation. Spectral analysis was accomplished

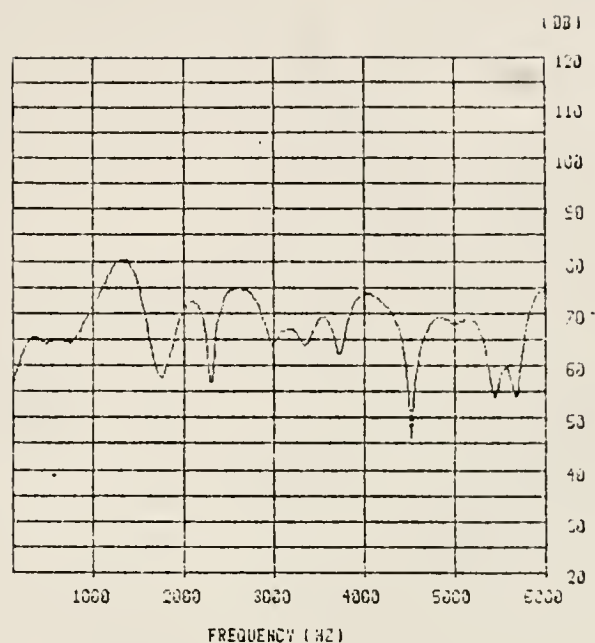
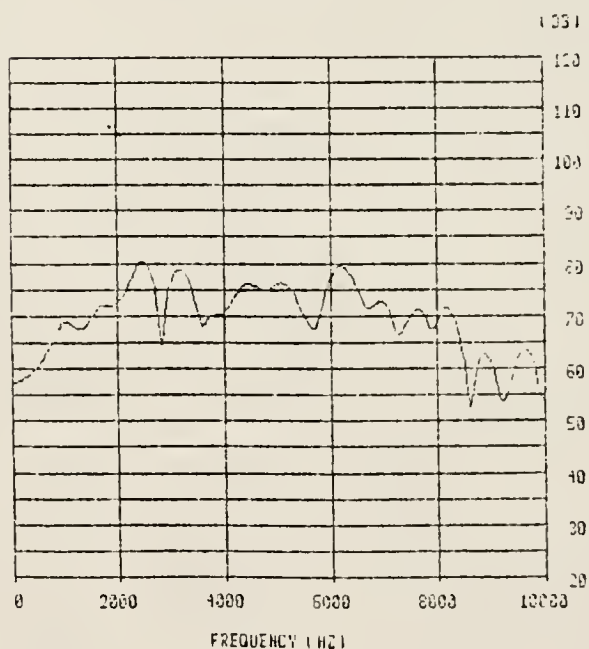
Industrial
NoiseBroadband
Noise

Figure 7. Fast-fourier transform (FFT) performed on industrial noise (top), and broadband noise (bottom) stimuli.

by coupling the Beyer DT48 earphone to a flatplate, attached to a standard 6cc cavity. Using a Bruel and Kjaer 1" microphone (type 4132), and amplifier (Bruel & Kjaer, model 2604), the signals were recorded on tape (Teac, X-10/X7), and fed into the IBM-PC. An ILS software package was used to perform the FFT analysis. The spectrum of the two stimuli were similar, except for a 5 dB peak present at 1200 Hz in the industrial noise signal.

Behavioral Hearing Threshold Measurements

All subjects were trained during a trial session to perform standard Bekesy tracking procedures. Stable tracings with midline deviations of no more than 3 dB, and excursions of less than 10 dB were required before continuing. A computer program controlled all stimulus parameters. Algorithms provide for acceptance or rejection of particular trials based on the above criteria.

Thresholds for pulsed tones (250 msec with 50% duty cycle) were established at discrete frequencies of 0.5, 1.0, 2.0, 3.0, 4.0, and 6.0 kHz prior to, following, and 24 hours after the noise exposure. Tonal stimuli were generated by a Hewlett-Packard 3311A function generator, and routed through Colbourn Instruments timing, gating, and digital logic circuitry. A programmable attenuator activated by a hand-held switch was controlled by the subject.

Signals were delivered to a single earphone (Telephonics TDH-39) with a standard (MX-41) cushion. Calibration of all stimuli were performed prior to, and following the collection of data.

Due to the rapid recovery of behavioral threshold following noise exposure, only one ear was tested during the recovery period. The left ear of all subjects was designated as the "test ear," and the right ear as the "non-test" ear for all threshold and AR measurements. Three measures of behavioral thresholds were completed during the post-exposure period. Pure tone thresholds were measured two minutes after cessation of the noise, and following the post-exposure measurement of AR activity (approximately 30 minutes after cessation of the noise). This was done to verify that the auditory system was still under the influence of TTS when the last AR measurement was completed. Recovery thresholds were again established 24 hours after the noise exposure.

Acoustic reflex measurements

Acoustic reflex measurements were completed in a sound treated booth (Industrial Acoustics) while in a sitting position. Prior to placement of the probe assembly, subjects were screened otoscopically to ensure no active pathology or excess cerumen was present that would prevent the recording of AR activity. Following otoscopic

inspection, tympanometry was performed to verify that ambient pressure of the middle ear system (point of maximum compliance) was within normal limits. AR threshold screening was also completed to ensure the presence of reflex thresholds of at least 85 dB SPL (broadband noise elicitor).

General instrumentation

Measurements of the middle ear were made with a Grason Stadler 1723 Middle Ear Analyzer (220 Hz probe tone). This device allows for the simultaneous recording of admittance (Y), and its sub-component, susceptance (B). Outputs of the instrument were fed into two separate Data Translation analog-to-digital channels connected to an IBM-PC. The voltage outputs were then evaluated with the assistance of computer programs specifically designed for AR analysis. All programs were written to allow for accurate measurement of AR parameters while adjusting for temporal characteristics of the instrumentation. Generally, the program averaged the first 600 msec of baseline voltage prior to the introduction of the eliciting signal. Threshold determination was made when the voltage change, caused by the elicitation of the AR, exceeded the baseline voltage by two standard deviations. Elicitor presentation levels were controlled by the experimenter, or by automated computer program which systematically increased or decreased

attenuator settings. The lowest intensity level that elicited an AR response was considered threshold. Other specific programs will be discussed in the following sections.

Volume estimates

Research indicates that corrections must be made for ear canal volume in order to obtain valid estimates of admittance at the lateral surface of the tympanic membrane (Block & Wiley, 1979; Margolis, 1981; Popelka, 1984). This measure depends on many factors including size of the probe tip, depth of insertion, and length of time in the ear canal (Margolis, 1981). Thus, admittance values (in mmhos) obtained at -350 daPa were used as estimates of ear canal volume (Moller, 1965; Shanks & Lilly, 1981). All acoustic reflex measurements were made at ambient pressure (point of maximum compliance) with the same test-ear/non-test ear paradigm previously described for behavioral threshold testing. The elicitation earphone was placed over the left "test ear," while the probe assembly monitored AR activity in the right "non-test" ear.

AR data collection

Measures of AR threshold, latency, and magnitude were obtained prior to, and following the industrial noise exposure for all thirty subjects. Measures of acoustic reflex adaptation, however, were assigned to groups of subjects, and are described in a later section.

Data acquisition could be completed by the experimenter with keyboard commands, or by programs allowing automatic data collection. In the automatic mode, the eliciting stimulus was initially presented at a level below anticipated AR threshold. The signal was then increased in 2 dB steps until a response occurred. If a response was present during the initial presentation of the eliciting stimulus, the attenuator was adjusted until no AR activity occurred. Once the initial response was identified, the attenuator automatically decreased the signal by 4 dB, and three successive trials occurred at that level. The intensity level was increased in 2 dB steps, and three presentations per intensity level were completed until the criterion for threshold was met (greater than 2 standard deviations of baseline activity). The lowest intensity level required to elicit this criterion-based response was considered threshold. The automated program provided a visual display of each trial as it was being collected. This allowed the experimenter to accept or reject a particular trial.

If the presence of artifacts rendered a particular trial uninterpretable, the trial was repeated.

Magnitude

Magnitude/intensity functions were obtained after threshold was established. Starting at threshold, the computer program automatically increased the elicitor intensity level in 2 dB steps (two presentations at each level), up to 12 dB sensation level (SL) with respect to AR threshold. The intensity function was continued with one presentation per 2 dB increment up to 104 dB SPL.

Magnitude of each acoustic reflex was computed in terms of admittance (Y). The phase angle with each acoustic admittance measurement was preserved for all computations (Block & Wiley, 1979). Therefore, vector and phase values of susceptance (B), and conductance (G) were maintained for calculations of admittance during the contracted, and uncontracted state of the AR. The corrected values for (Y) and (B) were stored to disk for later analysis. Calculation of conductance (G) was made using the following formula:

$$G = \sqrt{(Y)^2 - (B)^2}$$

Magnitude of the AR was obtained by subtracting the admittance of the middle ear prior to elicitation of the response, from the admittance during maximum acoustic

reflex contraction. Calculation of AR magnitude was accomplished by averaging the first 600 msec of baseline activity, and subtracting from it the average admittance between 1200 and 1600 msec as shown in Figure 8. Following termination of the eliciting stimulus, the program then averaged the corrected admittance values from 2100 through 2485 msec. This was used to represent the time period when the AR had returned to its quiescent state.

Latency

Four latency points and two slope functions were calculated for AR activity elicited at each stimulus presentation level. Figure 9 illustrates these six measurements. Latency 1 (L1) was defined as the time from the beginning of AR elicitation to 10% of the measured steady-state admittance change. Latency 2 (L2) was the time required to reach 90% of maximum admittance change in the AR response. Latency 3 and 4 are similar measurements used to describe the offset characteristics of the acoustic reflex. Offset latencies (L3 and L4) were defined as the time from the instantaneous termination of the initial admittance change to 90% and 10% of the steady-state admittance change, respectively. The rate of admittance change was also measured to further describe the temporal characteristics of the AR. Two linear regression models

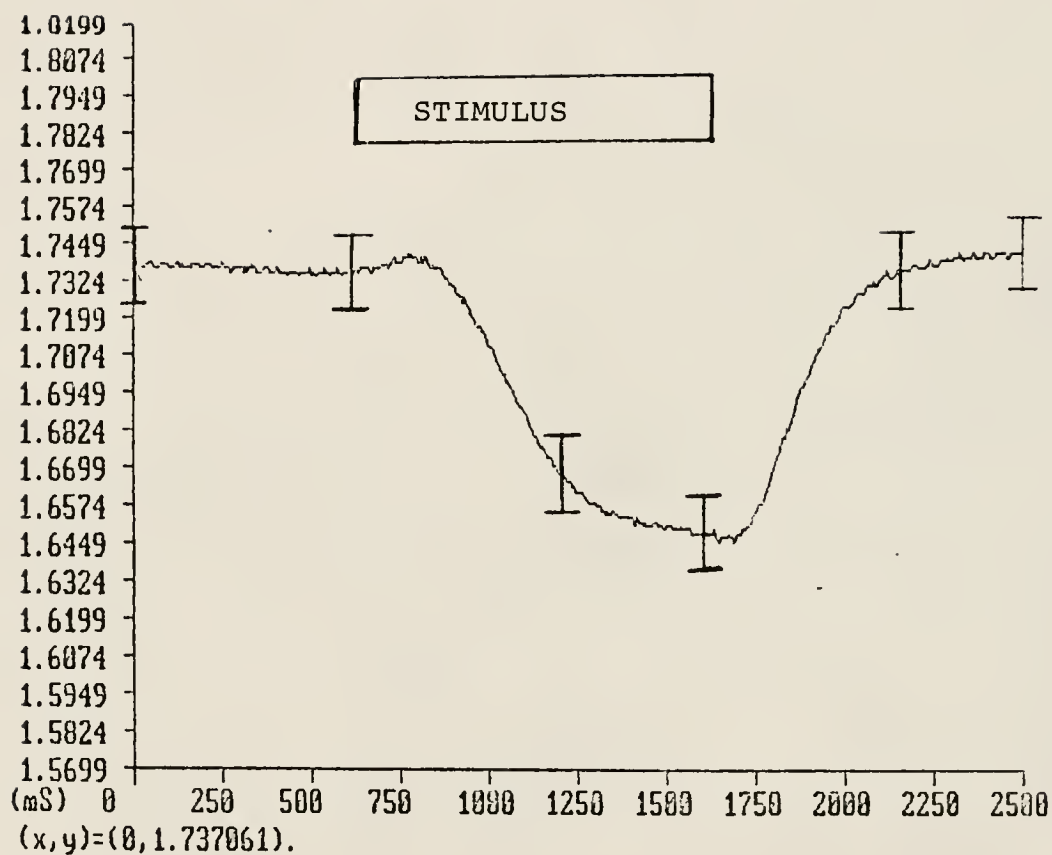


Figure 8. Example of computer averaging technique used to calculate onset and offset magnitude.

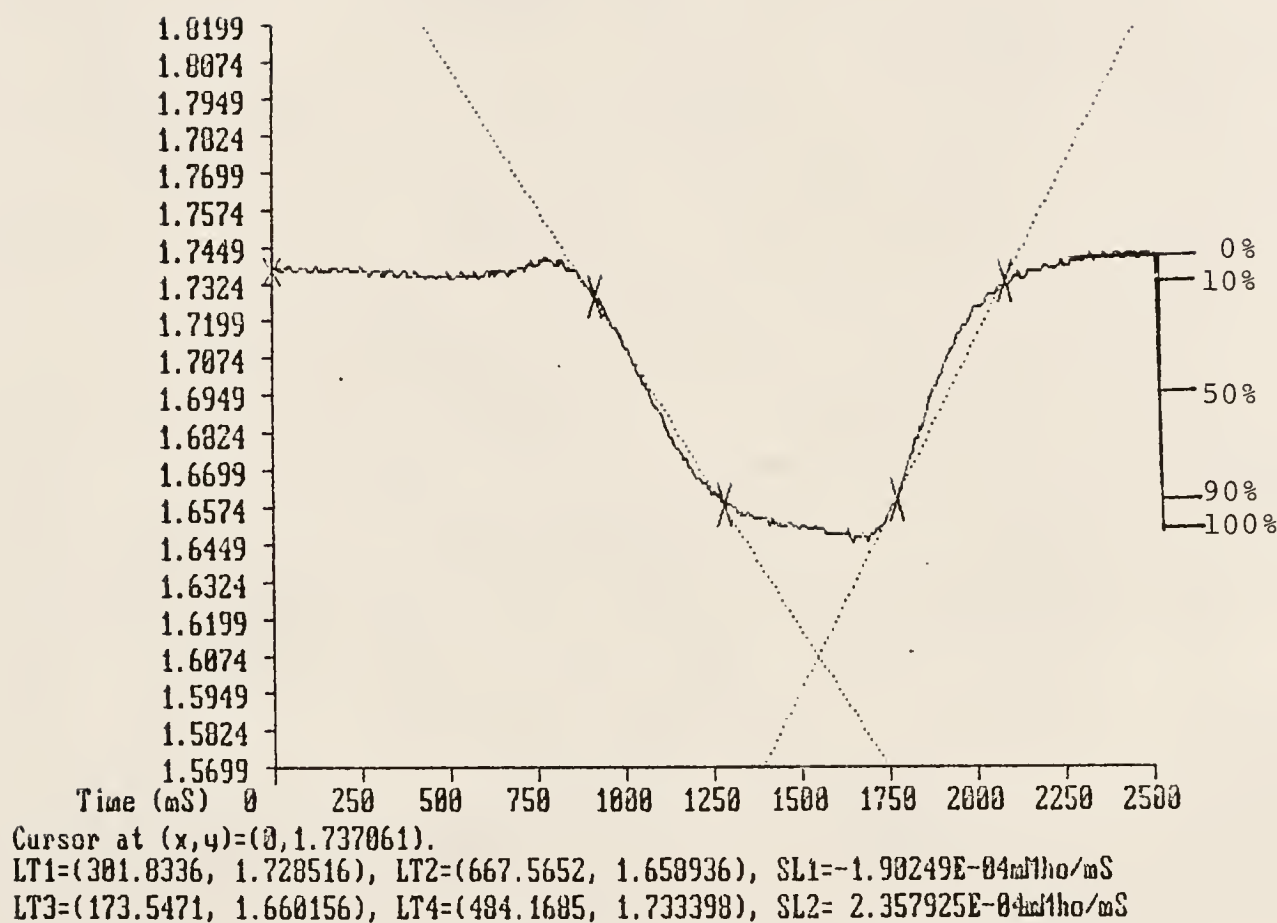


Figure 9. Example of four point latency (L1, L2, L3, L4) and slope functions used to analyze acoustic reflex activity. Latencies are expressed in msec and slopes are expressed as mmho change per msec.

were calculated from the initial and final transition of each response waveform. Slope 1 (S1) described the admittance change at the onset of the AR, and is expressed as the change in millimhos per msec from 10% to 90% of the steady-state admittance change. Slope 2 (S2) is a similar measurement at the offset of the acoustic reflex. It was defined as the change in millimhos per msec from 90% to 10% of the steady-state admittance value. Figure 9 shows the slope measurements for a particular AR response.

AR adaptation

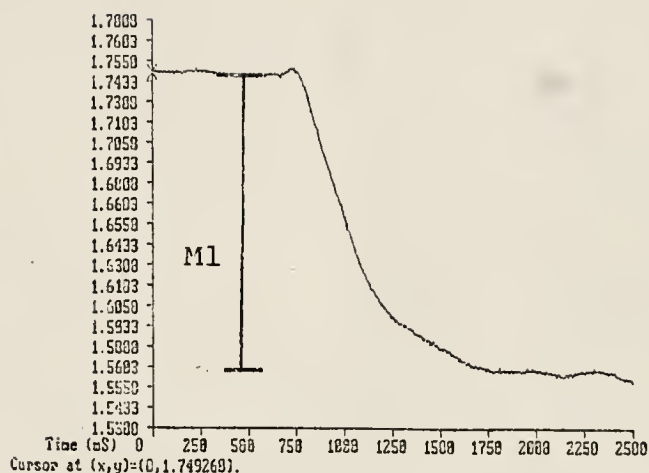
Several measures of perstimulus adaptation were evaluated in this study. Treatments were randomized in an effort to control for any possible order effect. Adaptation has been defined, as the decrease in AR magnitude over time for sustained and interrupted signals. One of the problems encountered when measuring AR adaptation, is that acoustic admittance constantly changes (Wilson, Steckler, Jones, & Margolis, 1978). Thus, the admittance measured at one point in time, may be different than the admittance measured at another point in time, even when those measures are completed on the same ear with no stimulus present (Wilson, Shanks & Lilly, 1984). In some individual cases, baseline drift may exceed total admittance change caused by acoustic reflex contraction. For this reason, measures of AR magnitude were

obtained at the beginning, and end of the sampling period only. Reflex adaptation was then expressed as percent change in AR magnitude (Y) over time as outlined by Wilson, Steckler, Jones, and Margolis (1978).

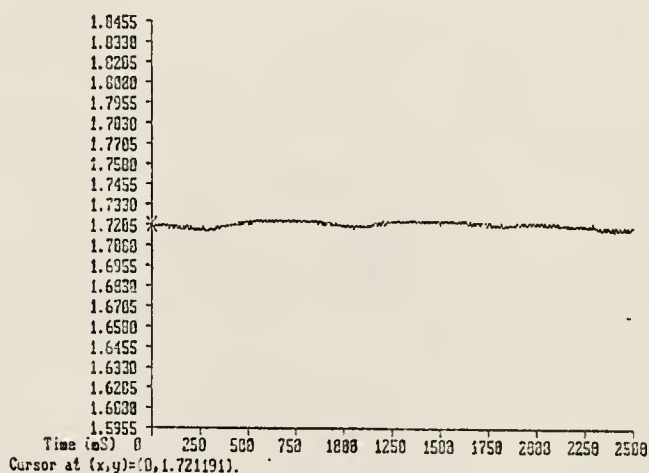
Measures of reflex adaptation for a broadband, and industrial noise stimuli were obtained by monitoring AR activity at 15 dB SL (re: AR threshold) for a period of 4.0 minutes. As previously mentioned, estimates of AR magnitude were obtained at the beginning, and end of this 4.0 minute period. Onset magnitude (M1), was calculated by subtracting the average baseline admittance (0-600 msec), from average admittance during maximum AR contraction (1200-1600 msec). At the end of the 4.0 minute period, a similar measure of offset magnitude (M2) was calculated. This value was determined by subtracting the admittance value just before termination of the eliciting signal (1000-1400 msec), from the admittance value after AR activity returned to its quiescent state (2100-2485 msec). Percent adaptation was then calculated from the admittance change observed at onset magnitude (M1) to offset magnitude (M2). Figure 10 provides an example of how AR adaptation was measured. The formula used to calculate percent adaptation is provided below.

$$\text{Percent Adaptation} = (1 - M2/M1) \times 100$$

STIMULUS
ONSET



30 seconds
into 4.0
minute
sampling
period



STIMULUS
OFFSET

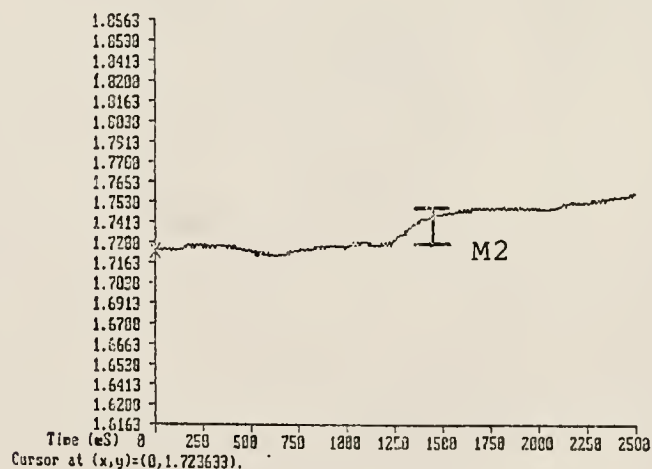


Figure 10. Standard adaptation for broadband noise stimuli. M1 represents onset magnitude, M2 represents offset magnitude.

Three measures of perstimulus adaptation were obtained during the pre-exposure session. Adaptation estimates were completed for a continuous broadband noise, continuous industrial noise, and broadband noise with specified interruption intervals. Signals used for the broadband elicitor were externally generated by a Colbourn Instruments noise generator, routed through a Colbourn Instruments rise/fall gate, mixer/amplifier and Hewlett-Packard 350D attenuator. Timing of stimulus presentation was controlled by Colbourn Instruments gating and timing devices interfaced with the microcomputer (IBM-PC). The stimulus used for the industrial adaptation series was the same as the fatiguing stimulus used in the 2.0 hour noise exposure. Tape output of a Panasonic 612 stereo cassette recorder was fed through the same Colbourn Instruments rise/fall gate, mixer/amplifier and Hewlett-Packard attenuator. Timing of the taped signal was accomplished by placing a pulse on the second (left) channel of the tape recording which initiated sampling activity of the microcomputer (IBM-PC). Eliciting signals were presented through a TDH-39 earphone with MX-41/AR cushion.

Standard adaptation for continuous broadband noise was completed by calculating percent adaptation in the fashion previously described (see Figure 10). An estimate of adaptation for industrial noise was also obtained in a similar manner (see Figure 11). Following the collection of these measures for continuous signals, other measures

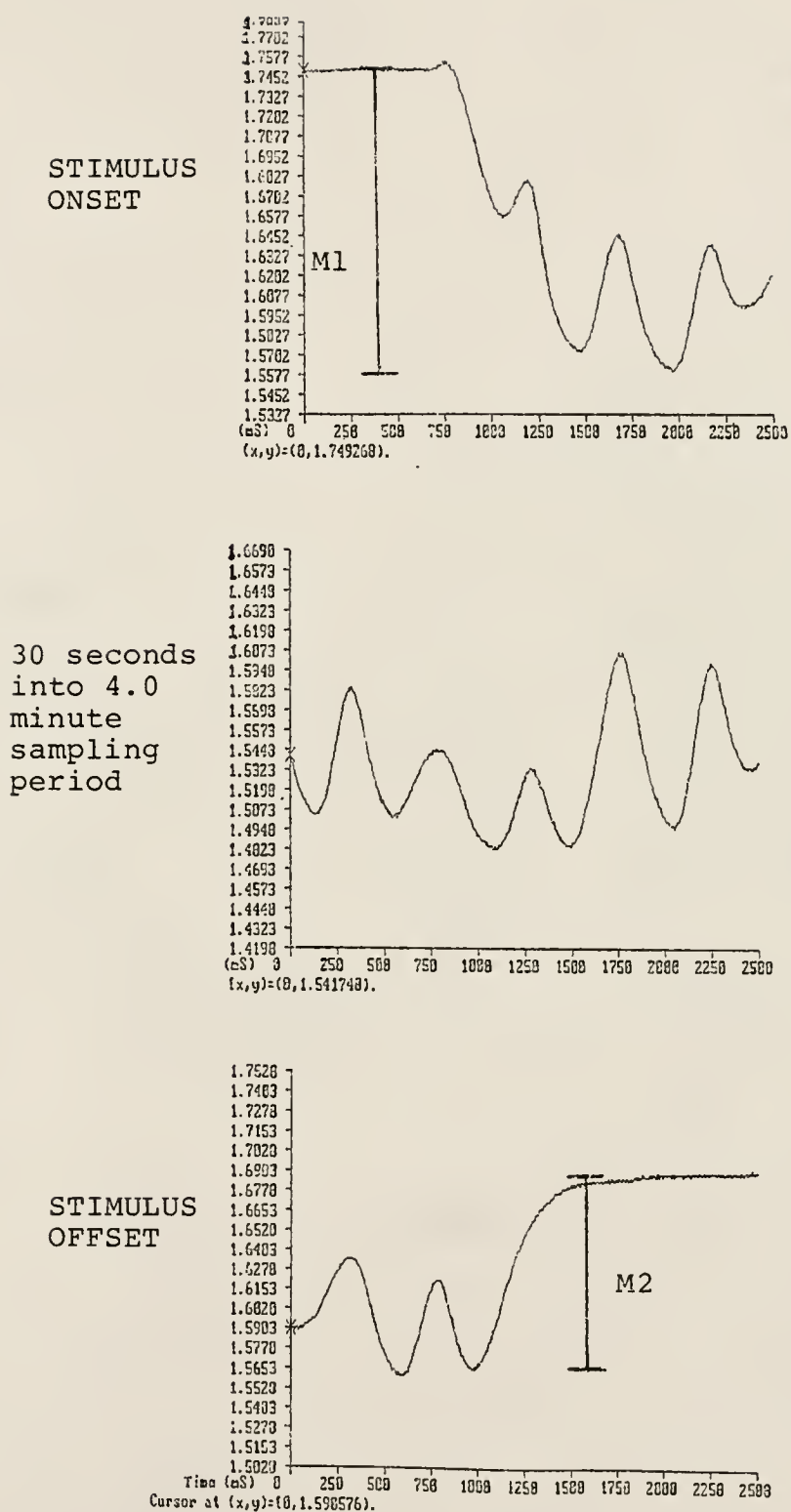
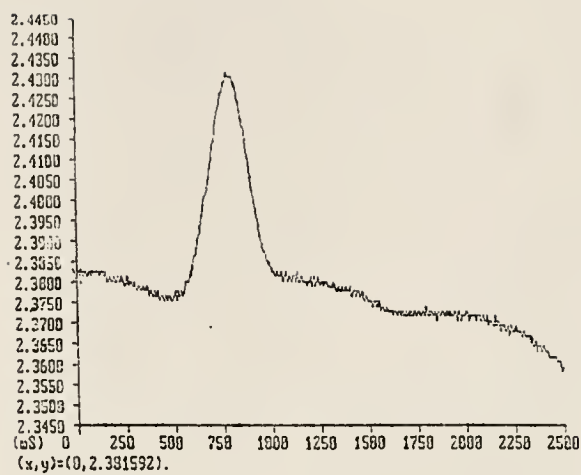


Figure 11. Example of industrial noise adaptation series. M1 represents onset magnitude, M2 represents offset magnitude.

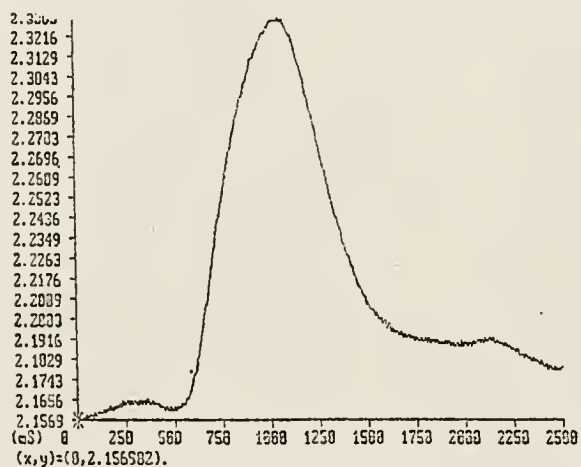
of AR function were obtained by briefly interrupting the sustained broadband signal. The signal was introduced at 15 dB SL (re: AR threshold), but was switched off, then on again, for specified interruption intervals of 250, 500, 750 msec (see Figure 12). For convenience, this new procedure was labeled the reflex interruption test (RIT). Ten subjects were subjected to one of the three different RIT treatments during the pre-exposure, and post exposure sessions.

One possible utility for the RIT would be in the evaluation of AR activity over time without the confounding influence of baseline drift. In this way, AR offset, and onset properties to a rapidly changing stimulus can be measured. Due to the unique nature of this response, AR activity under RIT conditions was evaluated in two separate ways. For comparative purposes, percent adaptation was calculated in a similar fashion as the continuous broadband and industrial noise signals (see Figure 13). Further analysis was then completed on the RIT procedure. By introducing interruptions in the eliciting signal, the RIT provides us with a relative measure of offset as well as re-elicitation activity of the acoustic reflex system. Figure 14 demonstrates this effect. R1 in that figure represents the change in middle ear admittance following cessation of the eliciting signal, while R2 results from a re-elicitation of AR activity due to re-introduction of the broadband noise. Magnitude of each phase of this response (R1 and

250
RIT



500
RIT



750
RIT

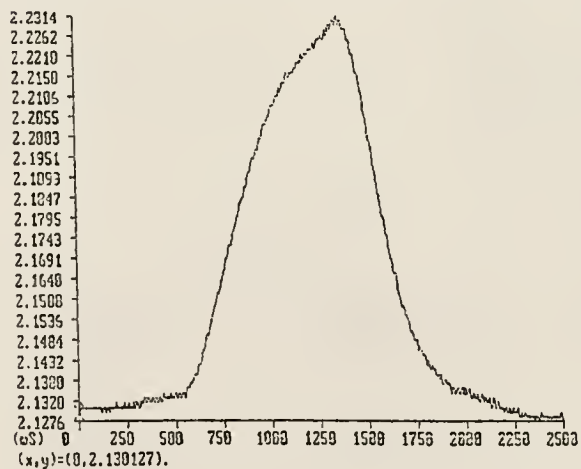
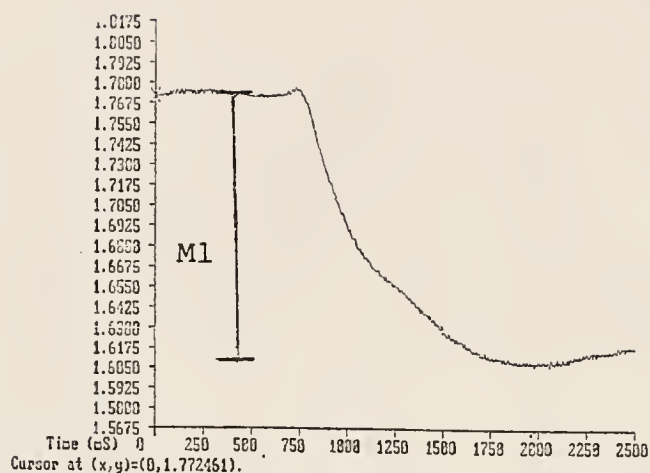
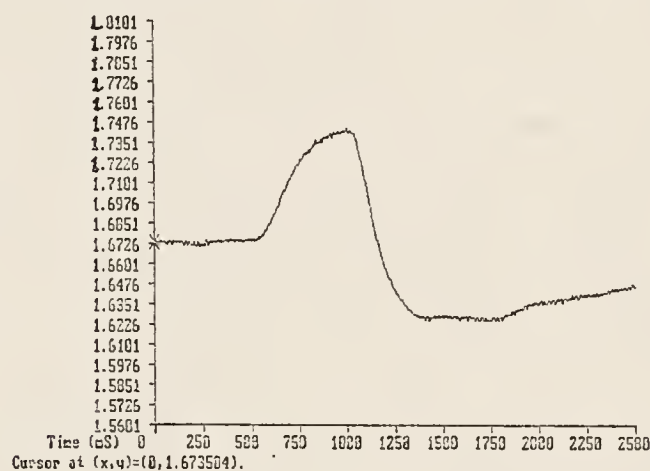


Figure 12. Example of 250 RIT (top), 500 RIT (middle), and 750 RIT (bottom) responses.

STIMULUS
ONSET



30 seconds
into 4.0
minute
sampling
period



STIMULUS
OFFSET

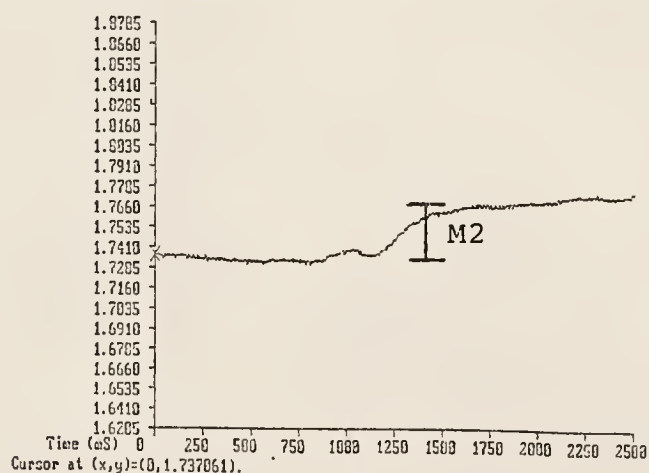


Figure 13. Reflex Interruption Test. M1 represents onset magnitude, M2 represents offset magnitude.

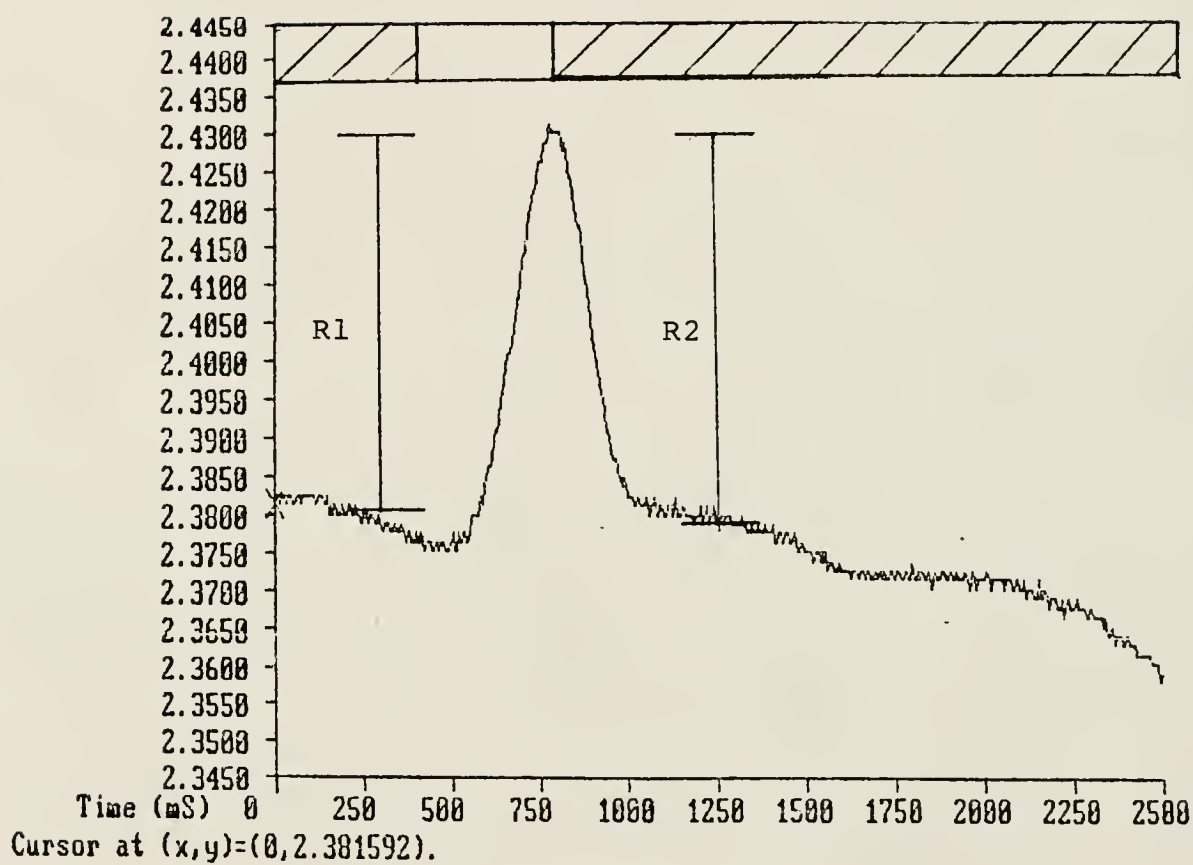


Figure 14. Example of RIT offset (R1), and re-elicitation (R2) magnitude.



Stimulus On



Stimulus Off

R2), was then evaluated over time for all stimulus conditions (250, 500, 750 msec).

Following the industrial noise exposure, standard adaptation, and RIT procedures were again completed. This was done in order to determine if changes in AR adaptation properties occur following industrial noise exposure. Measurement of these procedures were identical to the ones just described. Industrial noise adaptation was not completed due the rapid recovery of TTS observed during a pilot study utilizing an exposure of similar intensity and duration. It was felt that if significant changes were to be demonstrated in AR behavior following noise exposure, those measures would have to be completed while the auditory still under the influence of TTS. .

Calibration

Calibration of the Telephonics TDH-49 earphone with standard cushion (MX-41/AR) was conducted according to ANSI S3.7-1973 using a 1" Bruel and Kjaer condenser microphone coupled to a 6 cc cavity (type 4152). A Bruel and Kjaer pistonphone (Model 4220) was used to calibrate the microphone amplifier.

Different earphones were used for the noise exposure to provide comfort throughout the two-hour procedure. Circumaural cushions on the Beyer DT48 earphones, however, prevented calibration in the usual manner with a 6 cc

coupler. Thus, a flat plate coupler designed by Hepler (1984), was utilized according to procedures published by Michael and Bienvenue (1976). The flat-plate coupler is illustrated in Figure 15. This plate was designed to fit on top of the NBS 9-A coupler so that a flat surface was provided for the larger circumaural cushions. A vent made from catheter tubing was inserted between the Beyer earphone and flat-plate coupler. This was done to prevent an airtight seal between the coupler and earphone which previously created calibration difficulties.

General calibration of the Grason Stadler 1723 Middle Ear Analyzer was conducted according to manufacturer's specifications. The admittance output voltage was then calibrated by connecting the probe assembly to a variable-volume syringe. Voltage change per unit physical volume change was calculated, and included in the computer program used for AR analysis. The system was calibrated so that a 100 mv change in output voltage resulted from a 1 mmho change in measured admittance at 220 Hz. Another confounding variable which required special attention was response time of the bridge. Response time of the middle ear analyzer was calibrated according to procedures described by Wilson, Shanks, Jones, and Danielson (1982). As recommended, the diaphragm of a TDH-49 earphone served as the floor of calibration cavity. The face of the earphone was then covered with a wide speculum and sealed to the

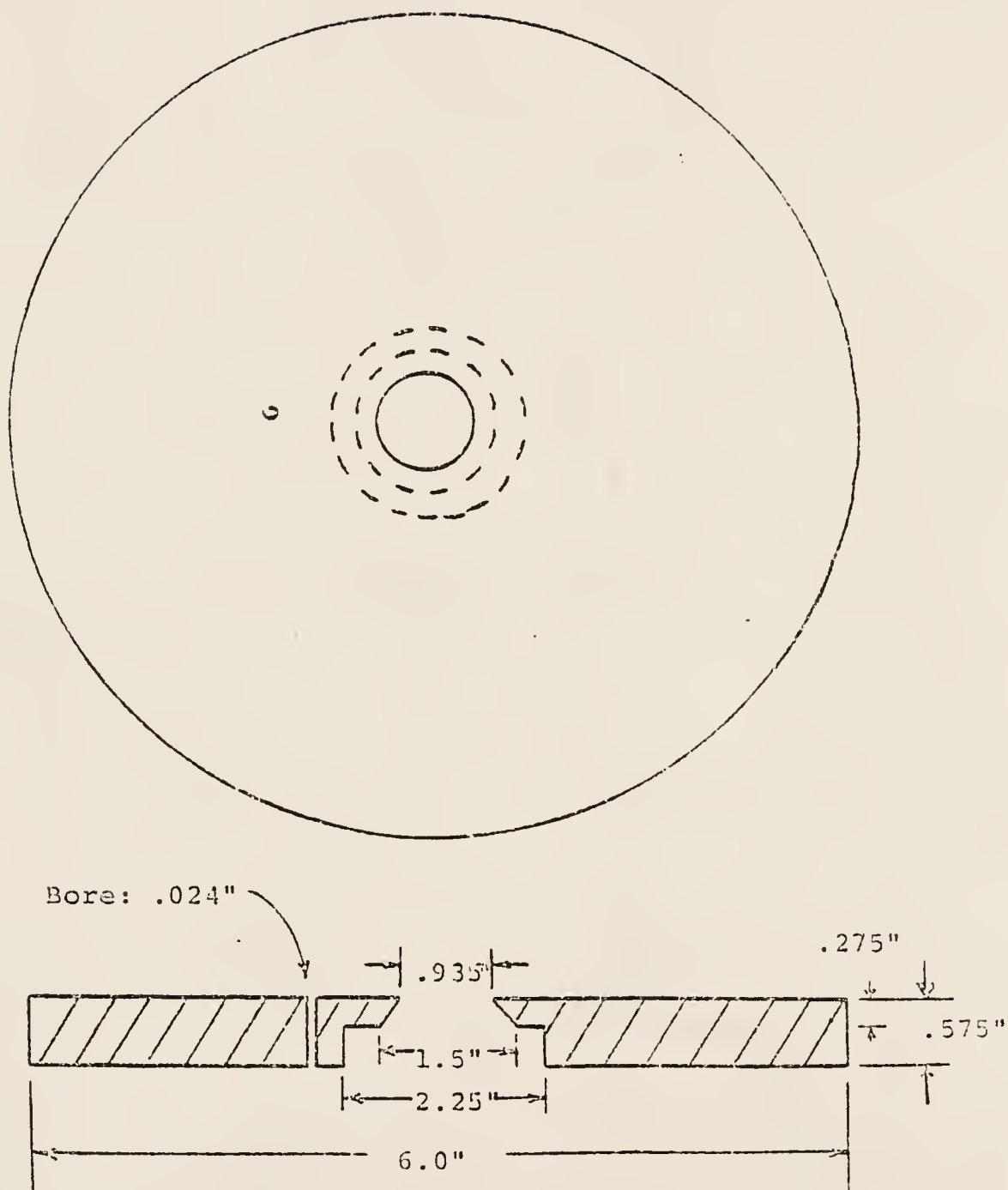


Figure 15. Flat plate used with the NBS 9-A coupler for Beyer DT 48 earphone calibration (after Hepler, 1984).

earphone. The earphone was then connected with tubing to a cut-off section of syringe. The probe tip of the GS 1723 was hermetically sealed to the syringe. With the 200 Hz probe tone present, an externally generated 1000 Hz tone was delivered to the cavity. The response time of the middle ear analyzer was then calculated (see Figure 16). As illustrated, the initial latency of the instrument was approximately 34 msec. Dwell time of the bridge at stimulus offset was estimated at 41 msec. These values were then entered into the computer program utilized in waveform analysis.

Data analysis

Statistical analysis were conducted at the Florida State University Computing Center utilizing an IBM 5520 computer. All computations were completed using Statistical Package for the Social Sciences (SPSS) software. Descriptive statistics for all threshold and AR parameters were obtained. Analysis of variance for dependent measures were completed according to Marks (1982) to answer the following experimental questions.

1. Do differences exist between pre-exposure and post-exposure values of the following response variables in normal hearing subjects after a two hour industrial noise exposure of 90 dB SPL?

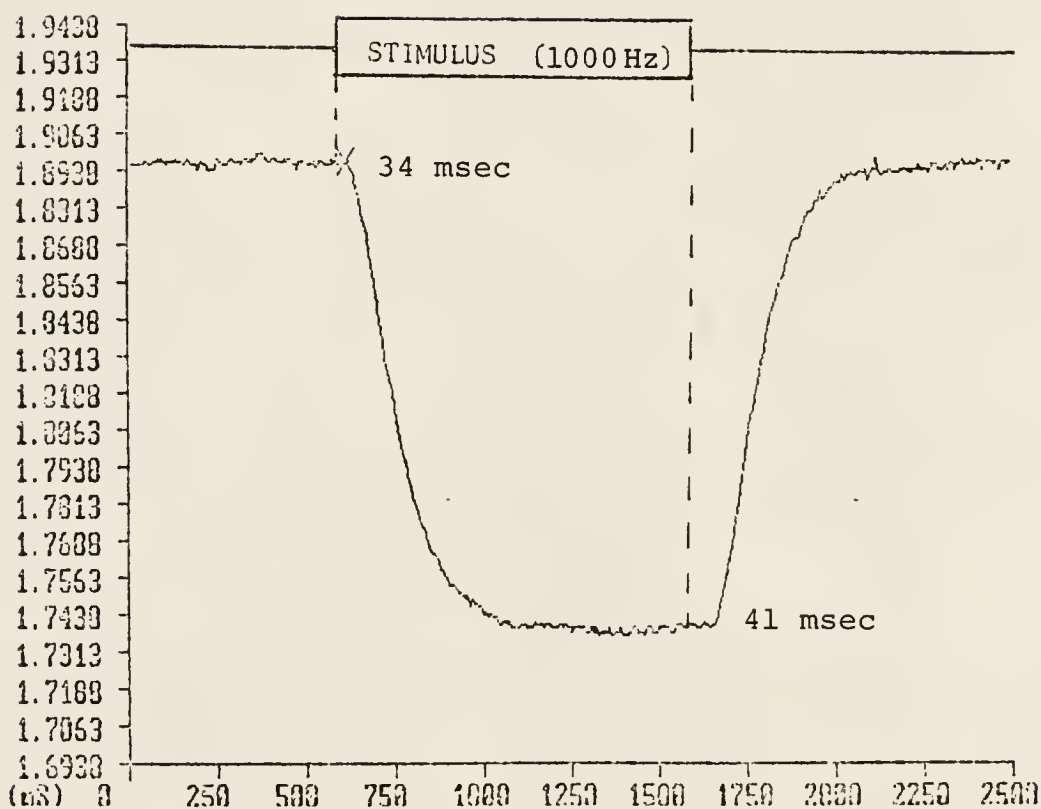


Figure 16. Analog output of a Grason Stadler 1723 middle ear analyzer when a test pulse is delivered to a calibration cavity.

- a) Behavioral thresholds at octave and half-octave intervals
 - b) Acoustic reflex threshold
 - c) Acoustic reflex magnitude
 - d) Acoustic reflex latency characteristics (onset and offset)
 - e) Acoustic reflex adaptation
- 2) Do differences exist in percent adaptation for the three stimuli evaluated in the pre-exposure session?
- a) Continuous broadband noise
 - b) Industrial noise
 - c) Interrupted broadband noise
- 3) Do any of these measures correlate to changes observed in audiometric behavioral thresholds following this same noise exposure?

A Tukey multiple comparison procedure was conducted whenever significant differences between treatments was obtained. Pearson Product-Moment correlations were utilized to investigate whether significant relationships existed between the various measures of AR activity and expressions of temporary threshold shift.

CHAPTER III RESULTS

Pre-Exposure Measures

Behavioral Thresholds

Behavioral thresholds were established utilizing Bekesy tracking procedures at octave, and half-octave intervals. Results show that thresholds were within the range expected for normal listeners. Average threshold values for pre-exposure testing were within 2 dB of ANSI (1969) standards at all test frequencies. As expected, greatest hearing sensitivity was demonstrated in the mid-frequencies (750-2000 Hz). Similar findings have frequently been reported in the literature (Shaw, 1974).

Thresholds were also established at the end of the experiment to verify complete recovery from the industrial exposure. Analysis conducted between pre-exposure and post-exposure thresholds, showed no significant differences present for the two sets of threshold measures (paired difference t-test). Mean recovery data were within 1 dB of pre-exposure thresholds, indicating complete recovery

at all test frequencies. Table 3 provides mean behavioral thresholds, and standard deviations for pre-exposure, and recovery sessions.

Acoustic Reflex Threshold

Acoustic reflex threshold was operationally defined as, the lowest elicitor level required to cause a shift in baseline admittance by at least two standard deviations (Block & Wiley, 1979; Hepler, 1984; Moul, 1985). Two responses out of three presentations at the same intensity level were required for final threshold determination. Thresholds were obtained for broadband, and industrial noise stimuli at the beginning of each experimental session. Descriptive statistics for the two elicitors are presented in Table 4. This table demonstrates a large range of threshold values for the two elicitors across subjects. Values reported are in general agreement with those of other investigators (Chun & Raffin, 1979; Gerhardt & Hepler, 1983; Moul, 1985). Also note, that average AR thresholds for the two elicitors are essentially the same. Acoustic reflex thresholds for the broadband, and industrial noise were 72.4 and 73.7 dB SPL, respectively. To verify this assumption, threshold data were subjected to a One Way Analysis of Variance for Repeated Measures (subjects X

Table 3.
Mean behavioral thresholds in dB SPL and
standard deviations for pre-exposure and
recovery data (N = 30).

Frequency (kHz)	Pre-exposure		Recovery		ANSI Standards dB SPL
	Threshold dB SPL (\bar{X})	Standard Deviation (SD)	Threshold db SPL (\bar{X})	Standard Deviation (SD)	
.5	13.5	5.4	12.8	6.5	11.5
.75	8.0	5.3	7.5	5.6	8.0
1.0	6.9	5.3	5.7	5.0	7.0
1.5	7.8	6.4	6.8	6.3	6.5
2.0	7.8	5.9	7.5	6.1	9.0
3.0	9.5	5.7	9.1	4.8	10.0
4.0	9.8	5.9	8.8	5.7	9.5
6.0	16.5	4.7	14.5	4.8	15.5

Table 4.
Descriptive statistics for acoustic reflex
pre-exposure thresholds (N = 30).

Elicitor	Minimum Threshold (dB)	Maximum Threshold (dB)	Mean (\bar{X}) (dB)	Standard Deviation (dB)
Broadband noise	60	88	72.4	8.2
Industrial noise	58	88	73.7	8.8

elicitor). Findings indicate that significant differences did not exist between the two elicitors at the $p = 0.05$ level.

Acoustic Reflex Magnitude

Magnitude of the acoustic reflex was obtained by subtracting the admittance of the middle ear prior to stimulus presentation, from the admittance value during maximum AR contraction. Research indicates that AR magnitude varies directly with intensity level of the activating stimulus (Wilson & McBride, 1978; Wilson, 1979; Silman, 1984). AR magnitude, along with latency, was obtained at several stimulus presentation levels (0-16 dB SL re: AR threshold). The plotting of changes in acoustic admittance as stimulus intensity increase, is typically called an acoustic reflex growth function.

AR growth functions were established following threshold determination for each subject. As expected, increases in elicitor intensity level resulted in growth of AR magnitude across subjects. Individual values of AR magnitude ranged from .001 mmhos at threshold, to .242 mmhos at supra-threshold levels. Figure 17 provides the average growth function of AR magnitude for thirty subjects. Also as previously reported, variability of AR magnitude between

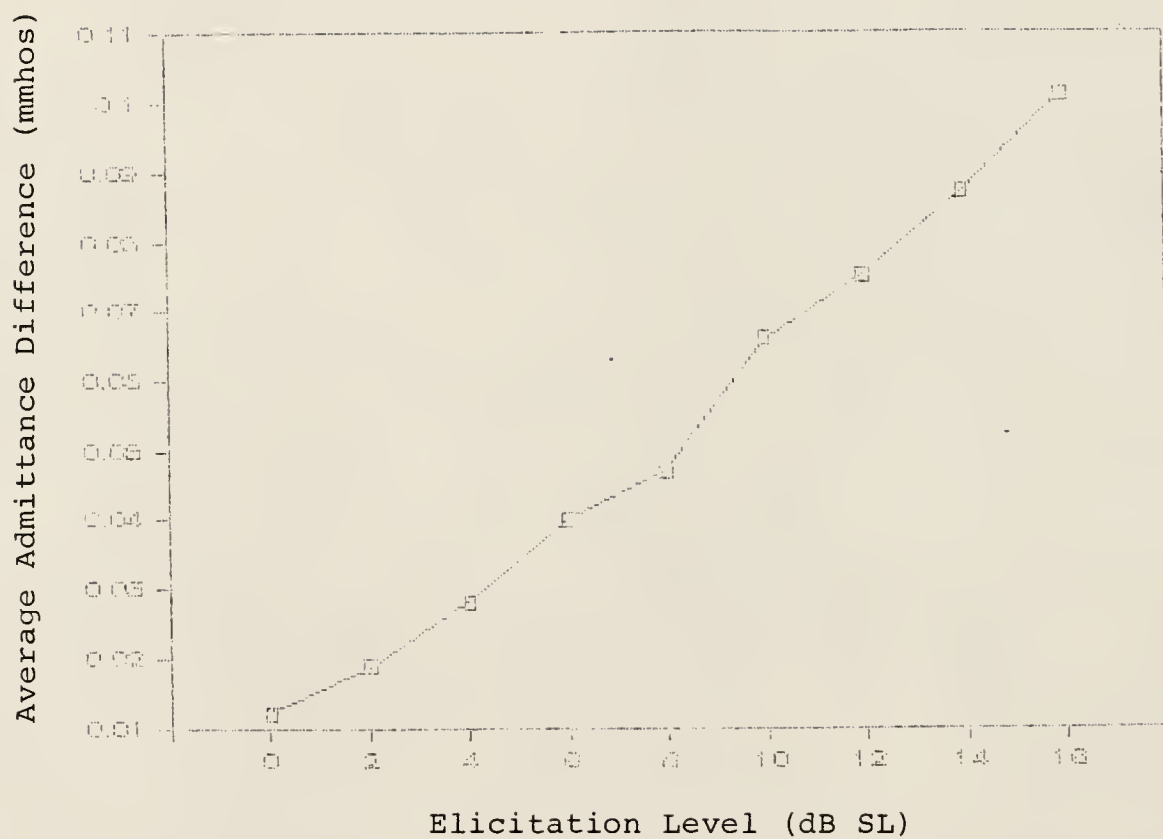


Figure 17. Average growth in AR magnitude measured in mmhos, per unit increase in elicitor presentation level for 30 subjects.

subjects was striking (Hepler, 1984; Moul, 1985). Figure 18 provides an example of AR activity for two subjects with similar thresholds stimulated by a 90 dB SPL broadband noise elicitor. As demonstrated, even under similar stimulus conditions, subjects varied considerably in magnitude of the acoustic reflex response.

Acoustic Reflex Latency

Temporal properties of the acoustic reflex system were also evaluated in this study. Four latency points and two slope functions for each stimulus presentation level were calculated (see Figure 9). Latency 1 (L1) was defined as the time in milliseconds from the beginning of an instantaneous admittance change to 10% of the steady state admittance change. Latency 2 (L2) was the time required to reach 90% of the maximum admittance change of the AR response. Latency 3 (L3), and Latency 4 (L4) were measures used to describe the offset characteristics of the acoustic reflex. They are defined as the time in msec, from termination of the eliciting signal to 90% and 10% of the steady-state admittance value, respectively. In an effort to further describe the dynamic properties of the AR, two additional slope functions were calculated. Slope 1 (S1), was used to describe the admittance change at the onset of the AR, and was defined as the change in millimhos per millisecond

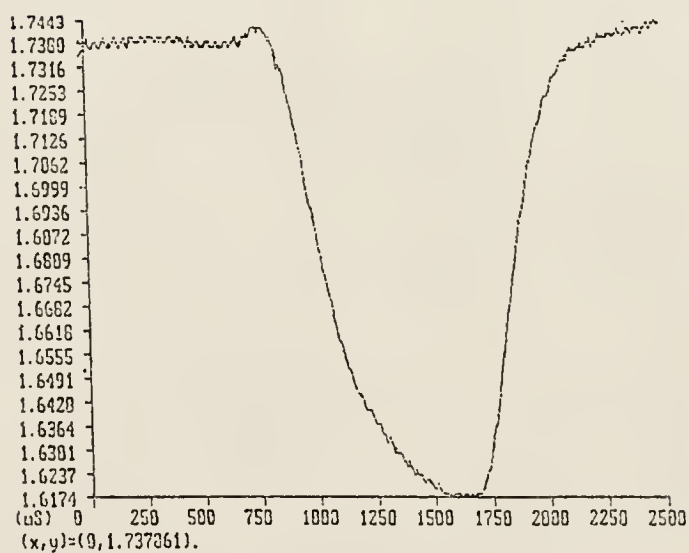
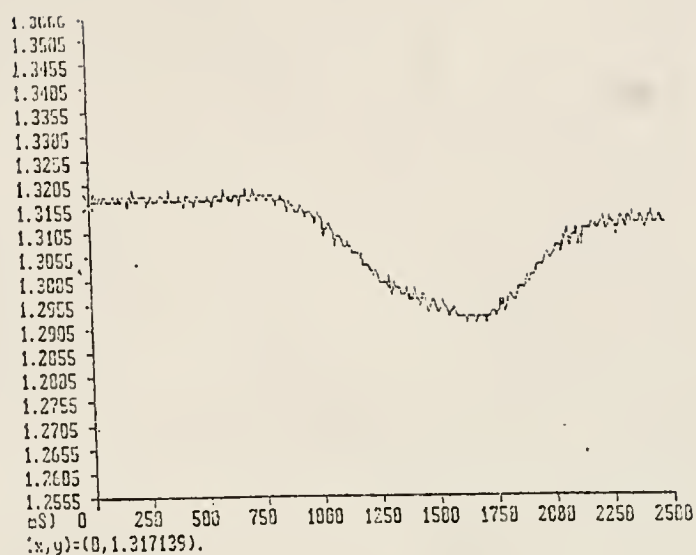


Figure 18. Example of AR magnitude difference between two subjects for the same elicitor and stimulus presentation level.

from 10% to 90% of the steady state admittance change. A similar measurement was made at the offset of the acoustic reflex. Slope 2 (S2), was calculated as the millimho per millisecond change from 90% to 10% of the admittance change following termination of the eliciting signal.

All measures of AR latency were completed at nine different stimulus presentation levels (0-16 dB SL re: AR threshold). This allowed for the evaluation of latency per unit increase in intensity level. Onset of the acoustic reflex response, measured by L1, showed a consistent decrease in latency per unit increase in intensity. In general, the greater the intensity level of the stimulus, the shorter the response time of initial AR activity (see Figure 19). This finding is consistent with the reports of other investigators (Borg, 1972; Dallos, 1973). The second measure of AR onset latency, L2, failed to display a similar relationship to stimulus presentation level. As illustrated in Figure 20, L2 values did not change considerably with increases in intensity level. The range of values for this measure was only about 40 msec. This would seem to suggest that stimulus intensity level has no effect on L2 measures.

Offset properties of the acoustic reflex system were evaluated by obtaining measures of L3, and L4. These values were defined as the time required by the AR system, to return to 90% and 10% of their respective steady state admittance value. Findings show that L3 behaved in a

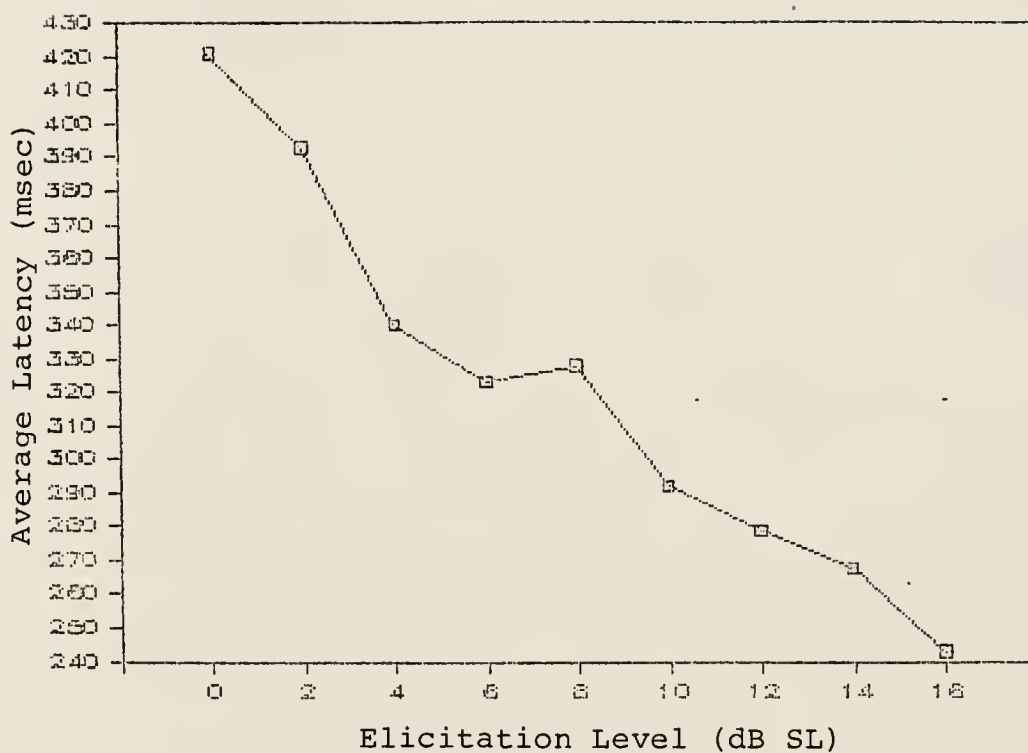


Figure 19. Average decrease in AR latency (L1) per unit increase in elicitor presentation level for 30 subjects.



Figure 20. Average change in AR latency (L2) per unit increase in elicitor presentation level for 30 subjects.

similar way to L1 by also demonstrating an inverse relationship to intensity. As illustrated in Figure 21, a consistent decrease in L3 latency was observed per unit increase in elicitor presentation level. The opposite was true for L4 latency values. In general, this measure showed an increase in latency, per unit increase in stimulus intensity level. In other words, as stimulus intensity increased, so did the value of L4 (see Figure 22).

Acoustic Reflex Adaptation

Five perstimulus adaptation procedures were evaluated during the pre-exposure session. Adaptation measures were obtained for continuous broadband noise (standard), continuous industrial noise, and three variations of the Reflex Interruption Test (RIT). All elicitors were presented at 15 SL (re: AR threshold). Acoustic reflex activity then was monitored over a 4.0 minute period. These five activators were evaluated in an effort to explain AR behavior for broad spectrum stimuli, possessing significantly different temporal properties.

Adaptation measures were initially normalized to allow for cross comparisons among subjects and stimulus conditions. The first 600 msec of admittance prior to stimulus onset was used to determine baseline characteristics of the response.

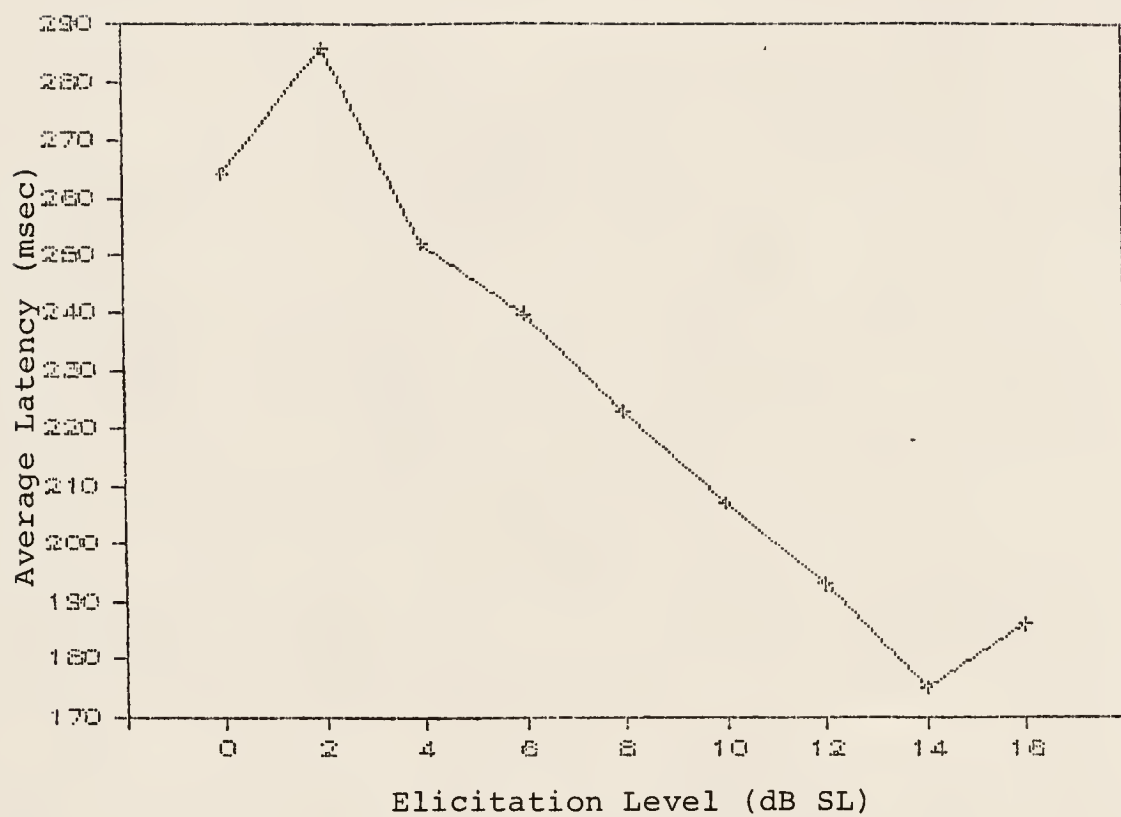


Figure 21. Average decrease in AR latency (L3), per unit increase in elicitor presentation level for 30 subjects.

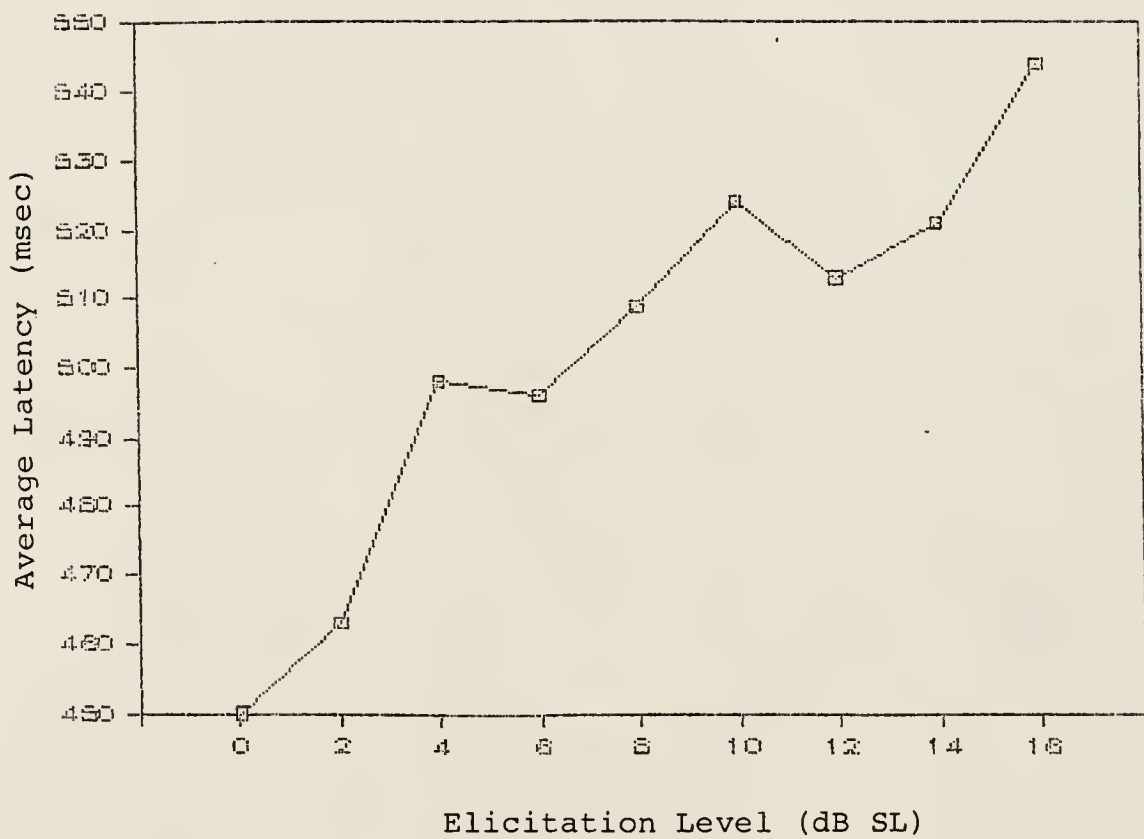


Figure 22. Average increase in AR latency (L4), per unit increase in elicitor presentation level for 30 subjects.

Onset magnitude (M1), was then calculated as the difference between this baseline admittance value and maximum admittance change observed during AR contraction (see Figure 10). This onset magnitude then served as the reference point for the adaptation series, and was given a value of 100%. Offset magnitude (M2) was obtained in a similar fashion at the end of the 4.0 minute period. Percent adaptation was then calculated from the admittance change observed from onset magnitude (M1) to offset magnitude (M2).

$$\text{Percent Adaptation} = (1 - M2/M1) \times 100$$

Because the Reflex Interruption Test represents a novel approach to the study of AR adaptation, preliminary analysis was completed to determine if significant differences exist in percent adaptation between the three RIT conditions (250, 500, and 750 msec). Results indicate that no significant differences or interactions were present for the various RIT protocols ($F = .04$, $p > 0.95$). This finding then allowed the three RIT procedures to be collapsed into one RIT treatment. This new grouping was then used for comparisons with the other stimulus conditions (continuous broadband noise, and continuous industrial noise).

This investigator then evaluated if differences exist in percent adaptation for the three stimulus conditions (continuous broadband noise, continuous industrial noise, and RIT) evaluated during the pre-exposure session? Findings indicate

that average adaptation for the thirty subjects was 77% for standard adaptation (continuous broadband noise), 61% across RIT conditions, and only 35% for the continuous industrial noise stimuli (see Figure 23). The adaptation treatments were then subjected to a One Way Analysis of Variance for Repeated Measures (subjects X treatments). Results showed that a significant main effect for elicitor condition was present ($F = 11.4$, $p < 0.01$). Post Hoc analysis utilizing the Tukey multiple comparison procedure, revealed that differences existed between the industrial noise elicitor, and the two broadband signals. Significant differences were not obtained between the standard adaptation, and RIT procedures.

Further analysis was then completed for the RIT procedures. By introducing short pauses in the eliciting signal, the RIT provides us with a relative measure of offset, as well as re-elicitation activity of the acoustic reflex. Figure 14 demonstrates this effect. R1, represents the change in middle ear admittance following cessation of the eliciting stimulus, while R2 results from a re-elicitation of AR activity due to re-introduction of the broadband signal. One possible utility of the RIT procedure may be in the study of AR activity over time, without the confounding influence of baseline drift. Initially, however, efforts must be made to observe, and describe AR behavior in response to interrupted signals. This was the basic intent of this study.

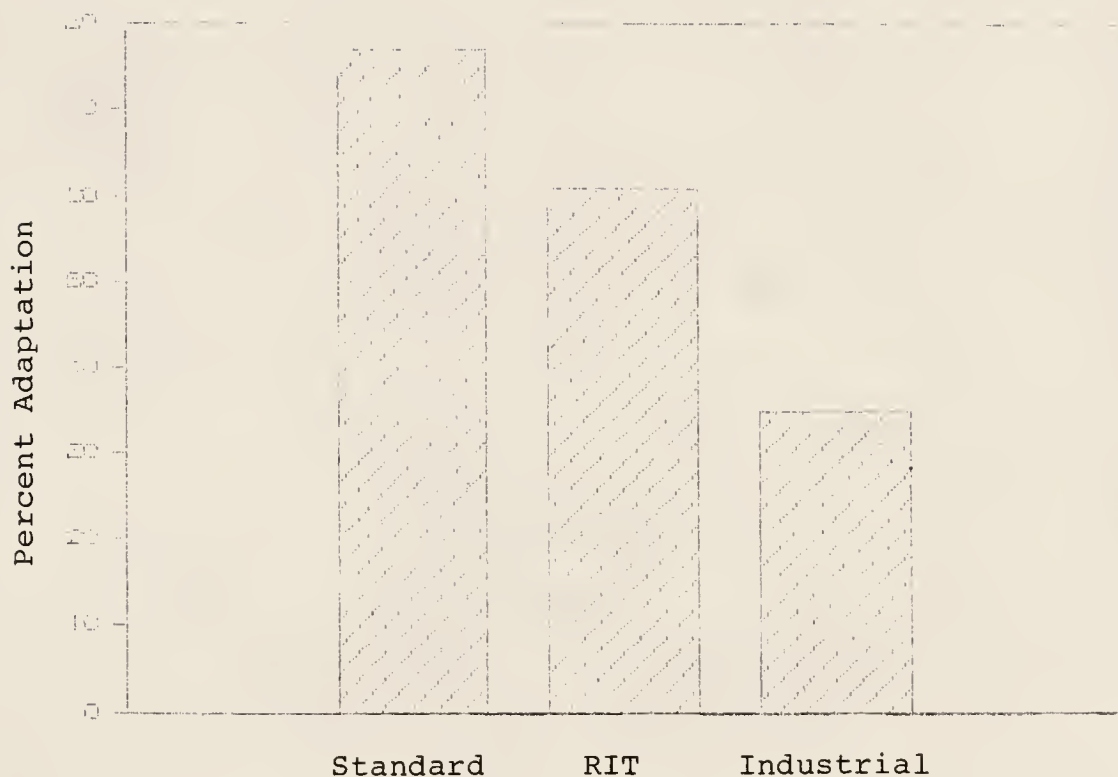
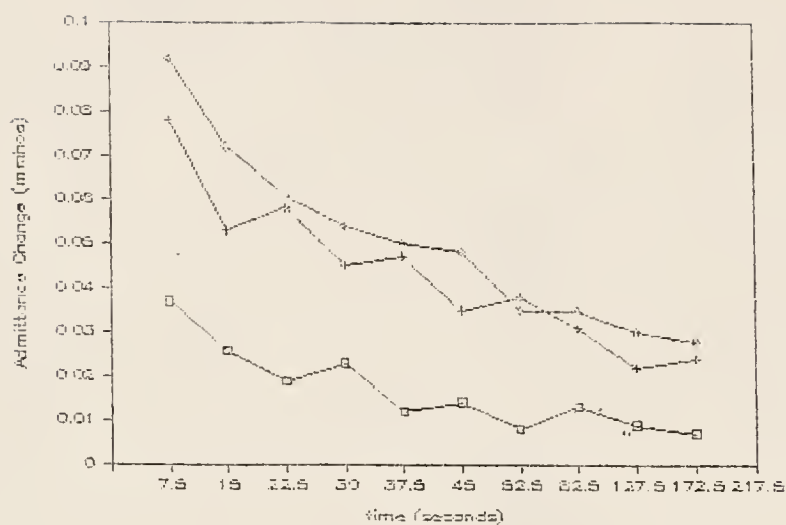


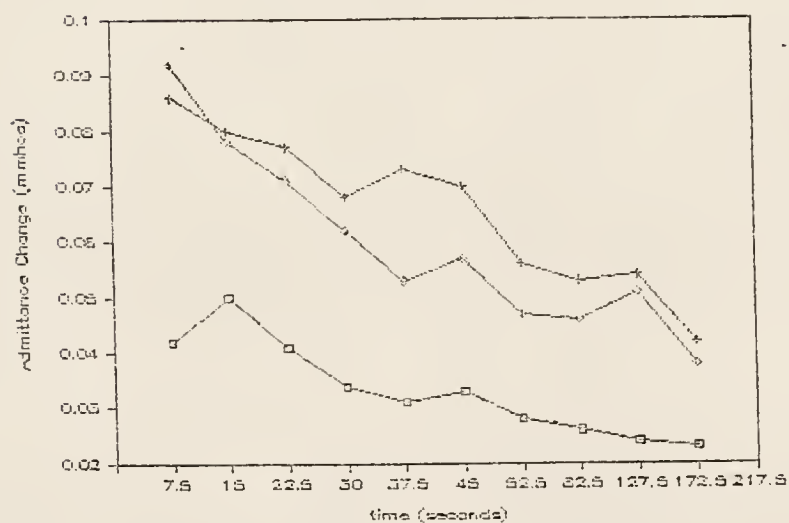
Figure 23. Percent adaptation at the end of a 4.0 minute sampling period for standard (continuous broadband noise), RIT (interrupted broadband noise) and industrial noise elicitors.

Measures of RIT magnitude (R1 and R2), were completed for all three RIT treatments. Magnitude of each response, recorded every 7.5 seconds, was calculated in the manner previously described. Findings indicate that, in general, the greater the interruption interval in the sustained signal, the larger the magnitude of the RIT response. RIT magnitude was greatest for the 500 and 750 msec procedures, with the 250 msec RIT demonstrating the smallest magnitude values (see Figure 24). It was also apparent, that RIT magnitudes were very similar for the 500 and 750 msec RIT conditions. In other words, similar RIT responses were obtained regardless if the broadband elicitor was interrupted for durations of 500 or 750 milliseconds. Comparisons of each phase of RIT behavior revealed that R2 (re-eliciation) magnitude, was consistently greater than R1 (offset) magnitude for all RIT treatments. This finding was true for both pre-exposure, and post-exposure conditions (see Figure 25).

Changes in RIT magnitude over time were also evaluated during the 4.0 minute sampling period. Results show that the RIT response declines quite rapidly. As illustrated in Figure 24, decline in RIT magnitude to 50% of its maximum amplitude occurs in less than one minute for all three conditions. The 250 msec RIT appears to decline to half its original value in approximately 35 seconds. The



R1 Magnitude



R2 Magnitude

Figure 24. RIT magnitude values R1 (top), and R2 (bottom) for three RIT treatments (250, 500 and 750 msec).

□ - 250

+ - 500

◇ - 750

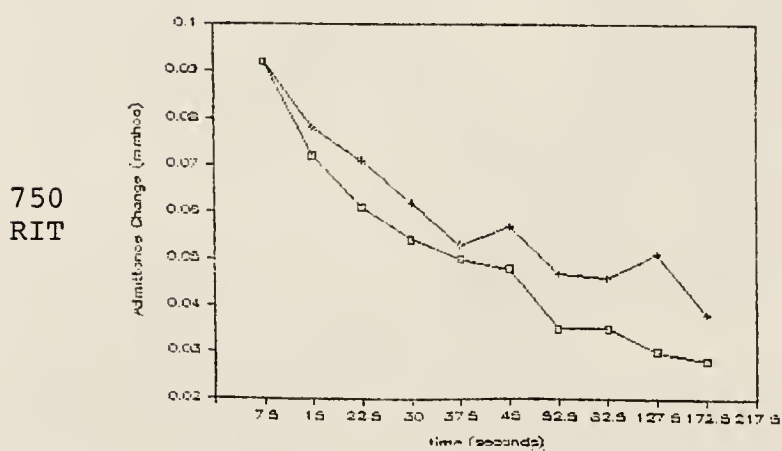
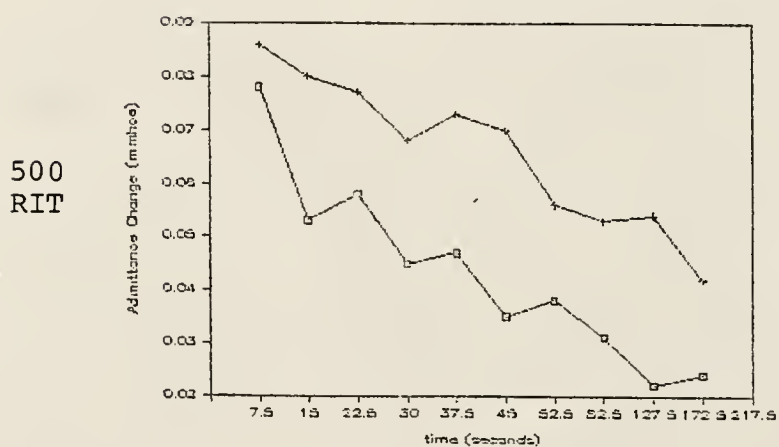
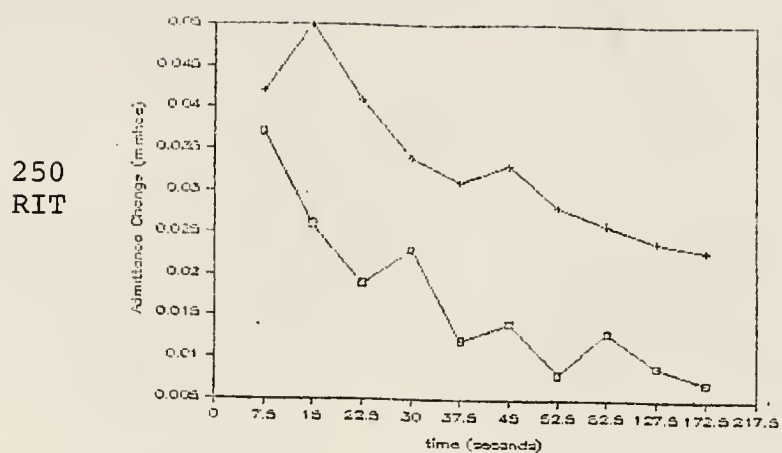


Figure 25. R1 vs. R2 magnitude for three RIT treatments.

□ - R1 magnitude

+ - R2 magnitude

longer interruption intervals (500 and 750 msec), decline to 50% of their maximum amplitude in approximately 50 seconds.

Effects of the Industrial Noise Exposure

Following the two hour industrial noise exposure, behavioral thresholds were established along with measures of acoustic reflex threshold, magnitude, latency, and adaptation. Changes in hearing sensitivity, and acoustic reflex behavior were then calculated by comparing pre-exposure to post-exposure measures. A review of the changes caused by the industrial noise exposure is presented in the following section.

Behavioral Threshold

A change in hearing sensitivity was observed following the two-hour industrial exposure of 90 dB SPL. Temporary threshold shift was calculated at each test frequency by subtracting the pre-exposure threshold, from the post-exposure threshold value. Results indicate that statistically significant threshold shift occurred for the test frequencies 1.5 through 6.0 kHz (paired difference t-test,

$p < 0.01$). Significant TTS was not obtained for the test frequencies 0.5 through 1.0 kHz. Figure 26 illustrates the TTS created by the industrial exposure measured two minutes after cessation of the fatiguing stimulus (TTS_2). Standard deviations are also provided. While average TTS was greatest at 3.0 kHz, individual data revealed TTS at other frequencies. Table 5 includes, mean, minimum, and maximum TTS values at each test frequency. In an effort to evaluate the effects of noise throughout the entire cochlear partition, TTS was also expressed as the sum of behavioral threshold shift across all test frequencies. This value was called Total TTS. Mean, minimum, and maximum values for this measure are also included in Table 5.

Following the collection of TTS_2 data, measures of acoustic reflex behavior were obtained. This process required approximately 30-40 minutes to complete. Estimates of behavioral thresholds were again established at the end of this AR testing period. This was done in order to verify, that the auditory system was still under the influence of TTS when the last AR measure was completed. Results of this second set of threshold measures (TTS_{30}) are illustrated in Figure 27. It is readily apparent when comparing this graph to TTS_2 data that recovery from the noise exposure was quite rapid. Findings do suggest, however, that the auditory system was still under the influence of TTS when the last AR measure was completed.

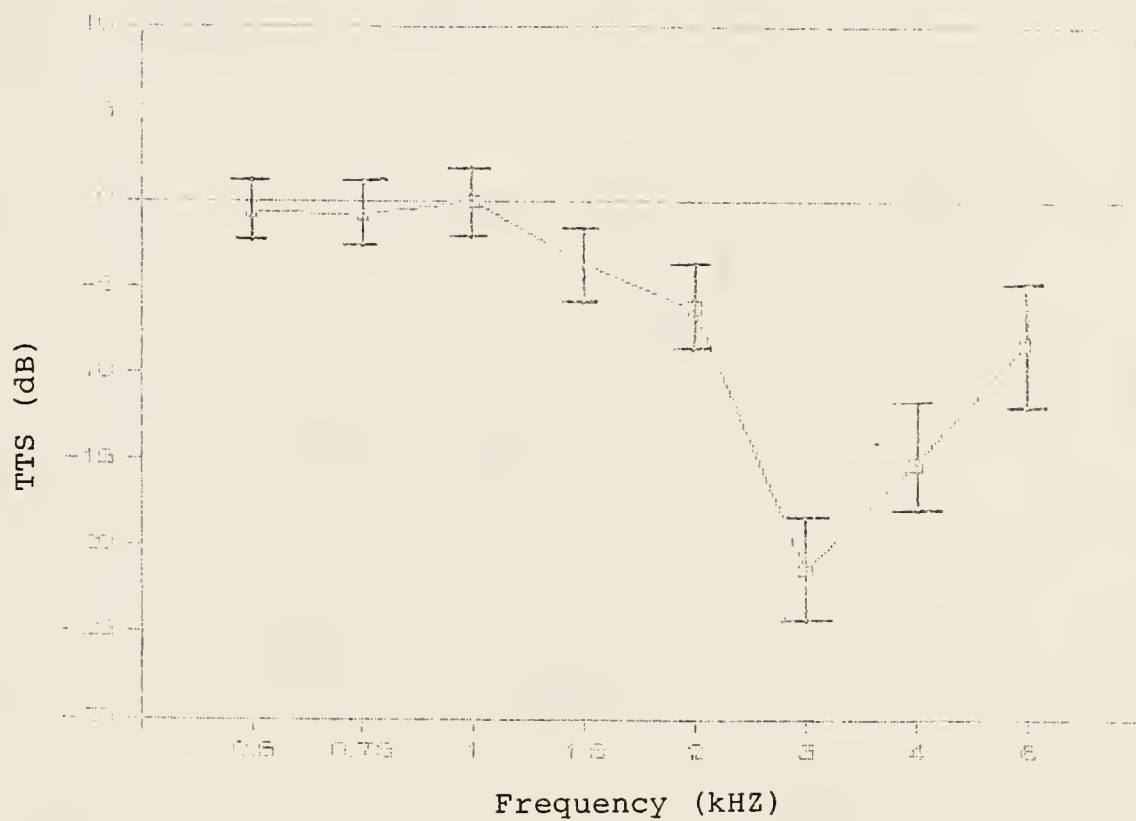


Figure 26. Mean TTS values by frequency for 30 subjects measured two minutes after noise exposure (bar = one standard deviation).

Table 5.
Descriptive statistics for expressions of temporary
threshold shift two minutes after industrial noise
exposure (TTS₂).

Expression of TTS (kHz)	Minimum Score (dB)	Maximum Score (dB)	Mean (dB)	Standard Deviation (dB)
Total TTS	15.4	104.5	56.2	21.9
.5	-6.4	6.3	.6	2.4
.75	-9.0	7.4	.8	3.4
1.0	-8.0	8.2	.1	3.5
1.5	-6.5	12.9	3.6*	4.5
2.0	-3.5	14.8	6.2*	4.4
3.0	10.2	31.5	21.4*	5.8
4.0	-1.9	25.9	15.2*	6.9
6.0	-6.7	22.1	8.3*	7.6

* $P < 0.05$

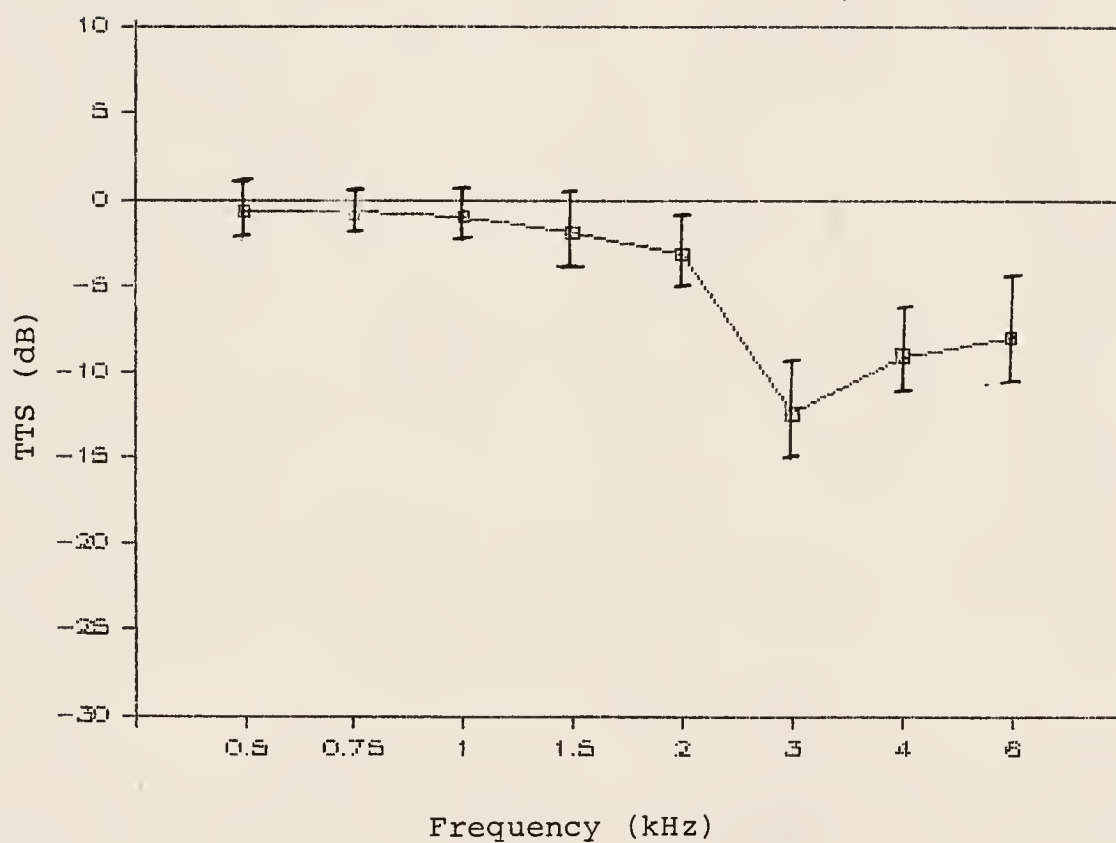


Figure 27. Mean TTS values by frequency for 30 subjects measured 30 minutes after noise exposure (bar = one standard deviation).

Statistically significant threshold shifts were still present for the same test frequencies demonstrating TTS two minutes after the noise exposure. Table 6 demonstrates this fact by providing descriptive information on TTS₃₀ values at each test frequency.

Acoustic Reflex Threshold

Acoustic reflex threshold measures were completed following noise exposure to determine if differences existed between pre-exposure and post-exposure values. Results indicate that significant threshold shift occurred following two hours of industrial noise exposure at 90 dB SPL ($F = 31.2$, $p < 0.001$). Table 7 presents descriptive information regarding AR threshold data collected during pre-exposure, post-exposure, and recovery sessions. This table also shows that on average, 5.46 dB of acoustic threshold shift was obtained. These findings are consistent with those of other investigators involving both animal and human models (Gerhardt & Hepler, 1983; Gerhardt, Melnick & Ferraro, 1979). After allowing 24 hours of recovery, AR thresholds were again measured. Analysis completed on thresholds collected during the pre-exposure vs. recovery conditions indicate no significant difference between average threshold values ($F = 2.9$, $p > 0.05$). The range of AR threshold shift also varied

Table 6.
Descriptive statistics for expression of temporary
threshold shift 30 minutes after industrial noise
exposure (TTS₃₀).

Expression of TTS (kHz)	Minimum Score (dB)	Maximum Score (dB)	Mean (dB)	Standard Deviation (dB)
Total TTS	-17.0	73.7	32.1	21.9
0.5	-18.9	4.7	-.64	4.0
0.75	-5.9	3.8	-.75	2.4
1.0	-8.6	5.8	-1.0	3.0
1.5	-9.4	9.3	1.9*	4.3
2.0	-6.2	11.3	3.2*	4.3
3.0	4.0	26.1	12.4*	5.1
4.0	-1.4	20.2	9.0*	6.4
6.0	-7.2	20.0	8.0*	7.1

*P < 0.05

Table 7.
Descriptive statistics for acoustic reflex
pre-exposure, post-exposure, and recovery
thresholds (N = 30).

Elicitor Condition (BBN)	Minimum Threshold (dB)	Maximum Threshold (dB)	Mean (X) (dB)	Standard Deviation (dB)
Pre	60	88	72.40	8.2
Post	62	88	77.86	8.9
Reflex Threshold Shift			5.46	
Recovery	58	86	71.20	8.5

considerably across subjects, from -4 dB to 18 dB SPL. Some subjects experienced a great deal of reflex threshold shift, while other threshold values didn't change at all. Figure 28 illustrates this finding.

Acoustic Reflex Magnitude

AR magnitude for nine separate intensity levels (0-16 dB SL re: AR threshold) were evaluated to determine if changes resulted from the two hour industrial noise exposure. Care was taken to control for any procedural variability between measurement sessions. It should be remembered, that all stimuli were presented in dB SL above AR threshold. Therefore, if reflex threshold shift occurred between pre-exposure, and post-exposure measures, elicitor intensity would be presented at a higher sound pressure across all nine presentation levels. Results under these conditions show no significant change in AR magnitude following noise exposure.

Acoustic Reflex Latency

One of the objectives of this study was to determine if differences in AR latency characteristics occur following a two hour industrial noise exposure. Results indicate



Figure 28. Reflex threshold shift for 30 subjects following a two hour industrial noise exposure at 90 dB SPL.

that significant changes did not occur for onset properties or slope functions at the $p = 0.05$ level. Changes were obtained, however, for offset properties of the acoustic reflex.

In general, delays in L3, and L4 were observed following the two-hour industrial noise exposure of 90 dB SPL. Closer study of L3 latency values reveal a 20 to 45 msec delay following noise exposure. These changes proved to be significant after being subjected to an Analysis of Variance for Repeated Measures ($F = 3.18$, $p < 0.05$). At high presentation levels, however, these differences were less apparent (see Figure 29). In fact, a significant interaction was obtained for L3 ($F = 15.0$, $p < 0.01$) across condition (pre vs. post), and intensity level (dB SL). In other words, although consistent delays in L3 were obtained for low to moderate presentation levels (0-10 dB SL), they were not observed at higher sensation levels (12-16 dB SL). Certain comparisons of pre vs. post exposure measurements even revealed a decrease in L3 latency following the industrial noise exposure.

The second offset parameter studied following industrial exposure was L4. This measure was defined as the time required to return to 10% of the steady state admittance value of the middle ear system. As with other measures of AR latency, pre-exposure vs. post-exposure values of L4 were compared to see if changes in latency characteristics

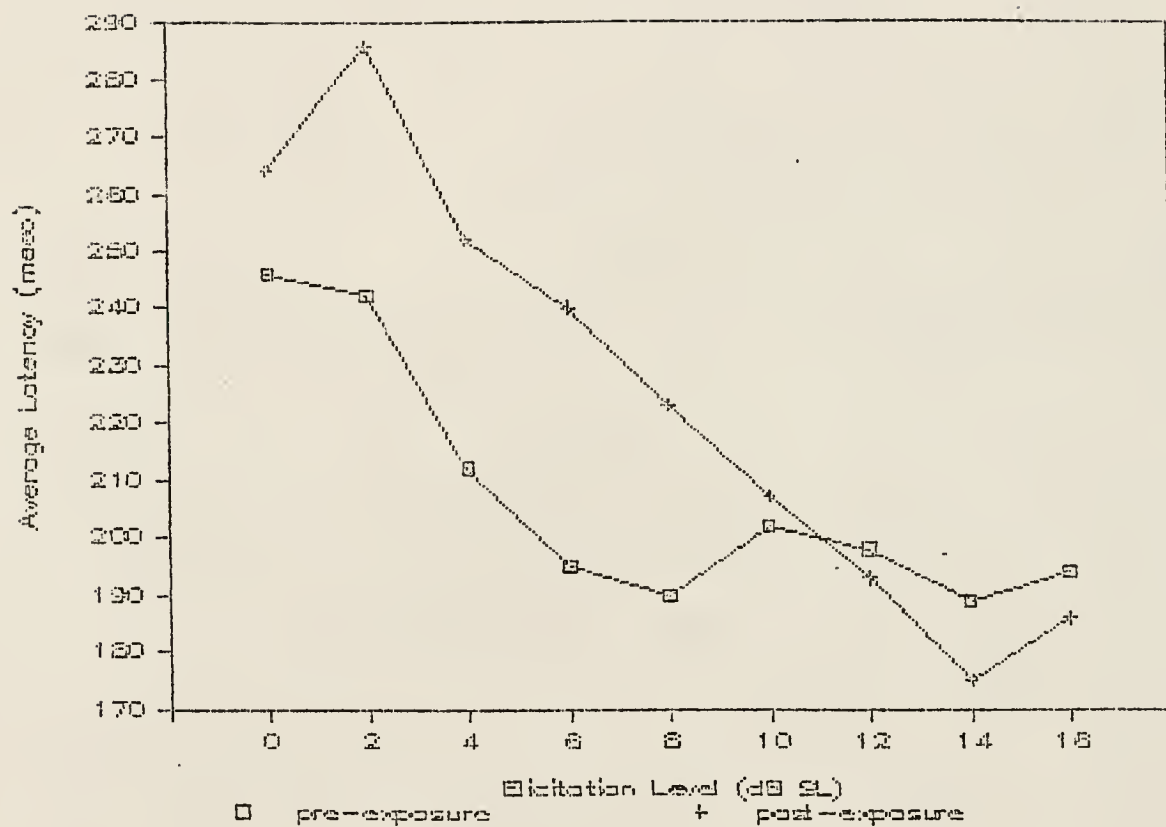


Figure 29. Average latency change for L3 per unit increase in elicitor presentation level (N = 30).

were affected by the noise. Results indicate that significant changes were present following noise exposure at all presentation levels ($F = 4.8$, $p < 0.01$). Average delays were between 25 and 55 msec across intensity levels, and are provided in Figure 30. No significant interaction was obtained for condition vs. presentation level as previously described for L3 values.

Acoustic Reflex Adaptation

Adaptation measures for continuous (standard adaptation), and interrupted broadband signals (RIT) were collected following the industrial noise exposure. It was felt that if significant changes were to be demonstrated in the adaptation process, those measures would have to be made while the auditory system was still under the influence of TTS. Because recovery from TTS created by this noise exposure was so rapid, a complete repeated measure design could not be conducted. Therefore, industrial adaptation measures were not collected.

This study then evaluated if changes in adaptation properties (as measured by percent adaptation) occurred following industrial noise exposure. Again, treatments were randomized in an effort to control for any order of presentation effect. Standard adaptation measures for the pre-exposure session were compared to post-exposure values.

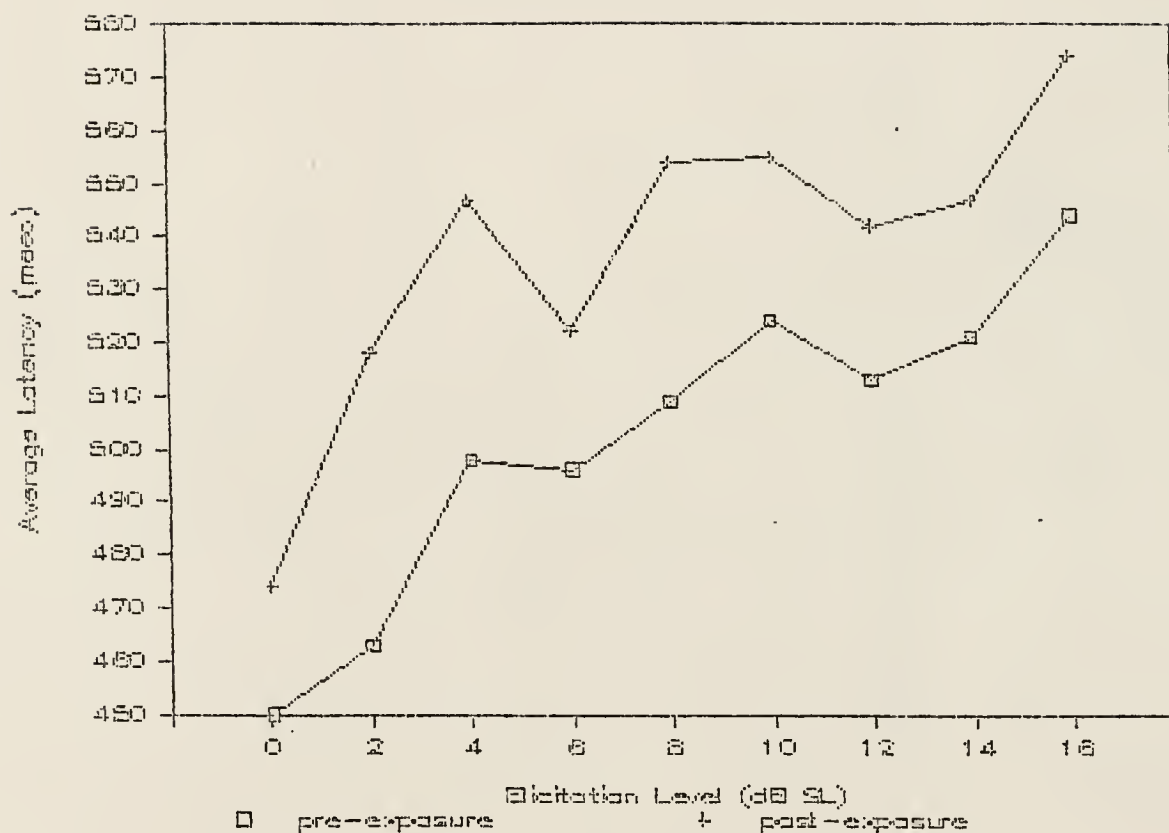


Figure 30. Average latency change for L4 per unit increase in elicitor presentation level (N = 30).

Findings indicate that significant differences did not occur between the two experimental sessions for this elicitor condition ($F = .39$, $p = 0.53$). Although not statistically significant, results did show a general increase in percent adaptation following noise exposure. Average adaptation was 77.4% for the pre-exposure session, and 80.6% for the post-exposure session.

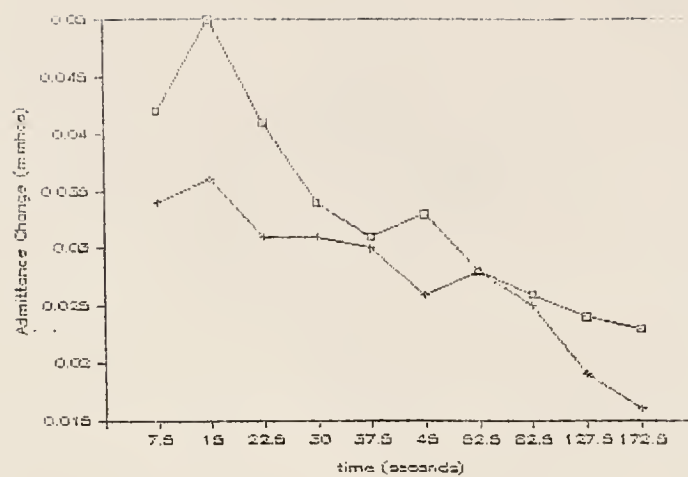
Analysis of RIT data revealed slightly different results. After comparing pre-exposure adaptation measures to post-exposure values, findings indicate that statistically significant changes were affected by the noise exposure ($F = 5.89$, $p < 0.05$). Results show that, on average, there was significantly greater adaptation following noise exposure (74.7%) when compared to the pre-exposure measures (61.0%). Table 8 provides descriptive information demonstrating this effect.

In an effort to further describe AR behavior under RIT conditions, individual magnitude values for each sampling period were measured. Recall, that RIT magnitude was expressed as two separate phases of the response. R1, which represented offset properties of the AR, and R2, which was thought to represent a re-elicitation of AR contraction. Both phases of this response were evaluated for the pre-exposure and post-exposure sessions. Findings indicate that significant changes did not occur in these measures following noise exposure as indicated by Figures 31 and 32.

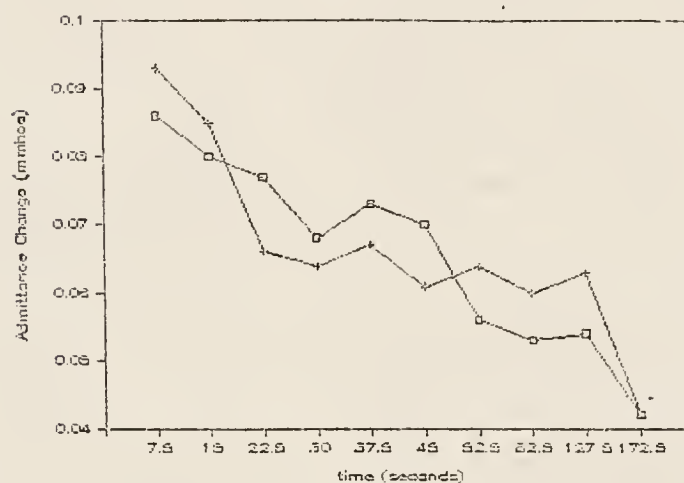
Table 8.
Percent decay values for RIT procedure during
pre-exposure and post-exposure sessions (N = 30).

Measurement Period	RIT Protocol (msec)		
	250	500	750
Pre-exposure	59.3%	63.6%	60.7%
Post-exposure	75.7%	70.0%	77.8%

250 RIT



500 RIT



750 RIT

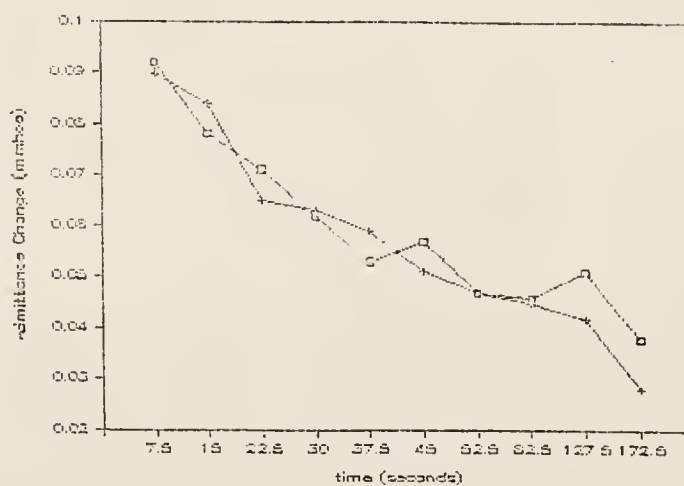
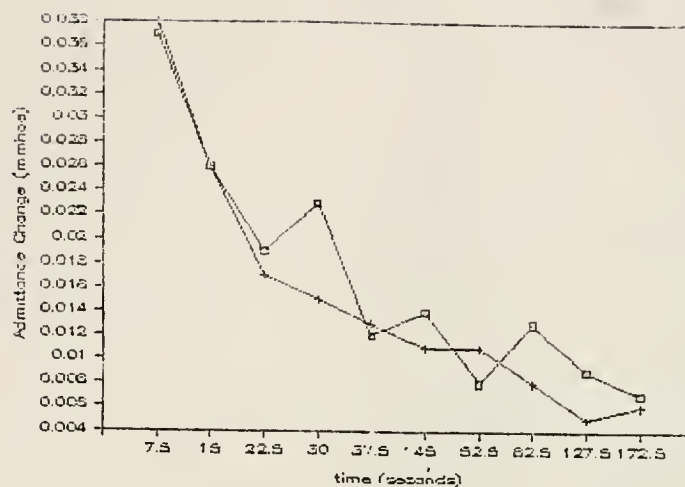


Figure 31. Pre vs. post-exposure R2 magnitude for three RIT treatments.

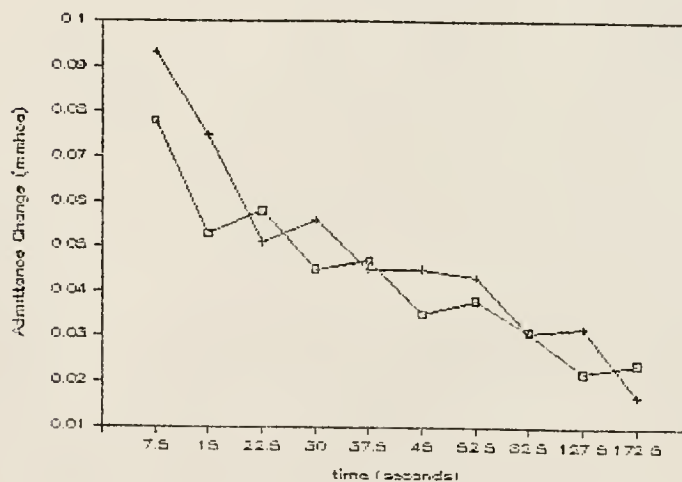
□ - pre-exposure

+ - post-exposure

250 RIT



500 RIT



750 RIT

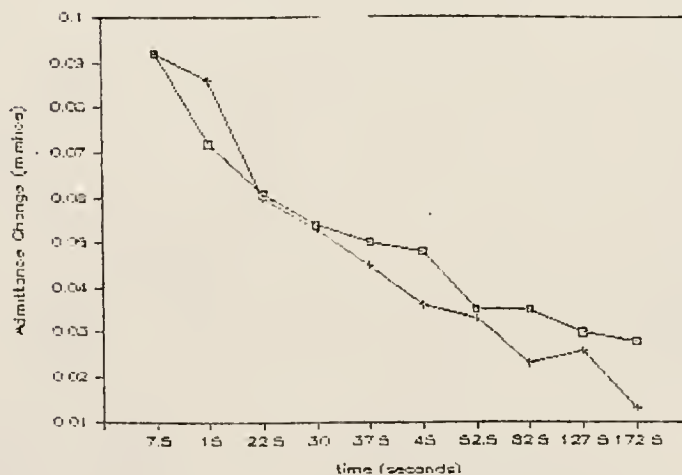


Figure 32. Pre vs. post-exposure Rl magnitude for three RIT treatments.

□ - pre-exposure

+ - post-exposure

Correlations

Potential relationships between measures of acoustic reflex behavior, and temporary threshold shift were also investigated in this study. Pearson Product-Moment correlations were conducted to determine if the measures of AR activity collected in this study could be used to partially explain the variability encountered in TTS.

Acoustic Reflex Threshold and Reflex Threshold Shift

Acoustic reflex threshold has previously been described as the lowest intensity level required to cause a shift in baseline admittance by more than two standard deviations. Expressions of TTS were correlated to AR threshold and showed no significant relationship between these two measures of auditory behavior. This finding is consistent with the reports of other investigators (Hepler, 1984; Moul, 1985). Reflex threshold shift (RTS), defined as the change in AR threshold following noise exposure, was also correlated to expressions of TTS. Results also indicate that no significant relationship exists between RTS, and measures of temporary threshold shift. Table 9 summarizes these results.

Table 9.
Correlations between AR threshold, reflex
threshold shift, and expressions of temporary
threshold shift.

Expressions of TTS (kHz)	AR threshold	Reflex threshold Shift
Total TTS	-.17	.18
0.5	.05	.17
0.75	-.12	.10
1.0	-.14	.20
1.5	-.05	.06
2.0	-.01	.05
3.0	-.23	.22
4.0	.01	.15
6.0	-.20	.02

Acoustic Reflex Magnitude

Potential relationships between AR magnitude and expressions of TTS were also investigated. Pearson Product Moment correlations between AR magnitude and indices of TTS are presented in Table 10. Results show that AR magnitude demonstrated a strong relationship to TTS created at 3.0 and 4.0 kHz. In fact, many of the correlations at these two frequencies were significant at the $p = 0.01$ level. Other frequencies did not display a similar relationship to TTS.

Acoustic Reflex Latency

Temporal characteristics of the acoustic reflex were also correlated to TTS. The four measures used to describe both onset and offset properties of the acoustic reflex were L1, L2, L3, and L4. The only measure of acoustic reflex latency that correlated to expressions of TTS was L4. This measure of AR offset response showed a consistent negative correlation to TTS created at 4.0 kHz. This relationship was significant at several presentation levels (see Table 11). Correlations of similar strength were not observed for other test frequencies. Table 11 provides a correlation matrix between L4 latency, and TTS at each test frequency.

Table 10.
Correlation matrix between AR magnitude and
expressions of temporary threshold shift.

Expressions of TTS (kHz)	0	2	4	6	8	10	12	14	16
Total TTS	-.22	-.19	-.15	-.05	-.22	-.04	-.07	-.01	-.01
0.5	-.07	-.21	-.21	.02	-.10	-.08	-.09	-.02	-.06
0.75	.24	.20	.19	.26	.14	.27	.21	.24	.19
1.0	-.07	.07	.02	.04	.03	.12	.00	.06	.06
1.5	.04	.24	.20	.22	.20	.38	.28	.36	.32
2.0	.01	.06	.18	.17	.11	.23	.23	.26	.27
3.0	-.51**	-.54**	-.40*	-.47**	-.52**	-.43*	-.37*	-.38*	-.29
4.0	-.46**	-.59**	-.57**	-.32	-.54**	-.50**	.47**	-.43*	-.40*
6.0	.10	.17	.09	.12	.02	.16	.13	.15	.13

** $p < 0.01$

* $p < 0.05$

Table 11.
Correlation matrix between AR latency (L4) and
expressions of temporary threshold shift.

Expressions of TTS (kHz)	AR latency (L4) elicited by different presentation levels (db SL re: **)	0	2	4	6	8	10	12	14	16
Total TTS		-.38*	-.16	-.22	-.26	-.10	-.13	-.05	-.20	-.03
0.5		-.26	-.07	-.37*	-.19	-.22	-.19	-.21	.08	-.06
0.75		-.12	.15	-.16	-.19	-.09	-.01	.13	.30	.15
1.0		-.01	-.01	-.15	-.04	.20	.00	-.15	.08	.31
1.5		-.07	-.16	.05	.09	.02	.02	.13	.04	.10
2.0		-.34	-.19	-.10	-.08	.00	-.02	.14	.24	.13
3.0		-.38*	.02	.04	-.04	.09	-.06	-.08	.12	.08
4.0		-.53*	-.37*	-.31	-.35	-.39*	-.43*	-.31	-.20	-.38*
6.0		.01	.01	-.09	-.23	-.01	.12	.13	.32	.06

* $p < 0.05$

**AR threshold

Adaptation

Measures of AR adaptation for the three eliciting signals (continuous broadband noise, interrupted broadband noise, and industrial noise) were also correlated to TTS. Findings indicate that no significant relationship exists between any measure of AR adaptation and temporary threshold shift. Table 12 provides a complete summary of these findings.

In summary, few measures of acoustic reflex behavior showed a significant relationship to expressions of TTS. As previously reported, AR magnitude appears to be significantly related to temporary threshold shift at frequencies maximally affected by the noise exposure. Offset latency L4, also appears to be related to at least one expression of TTS (4.0 kHz). Other measures of acoustic reflex behavior, namely AR threshold, onset latency, and adaptation, failed to demonstrate significant correlations to measures of temporary threshold shift.

Table 12.
Correlations between measures of AR adaptation
and expressions of temporary threshold shift.

Expressions of TTS (kHz)	Standard (Continuous BBN)	RIT (Interrupted BBN)	Industrial (Continuous)
Total TTS	-.16	.05	.12
0.5	-.29	-.21	.07
0.75	-.26	-.23	-.04
1.0	.20	.26	-.02
1.5	.17	.23	.06
2.0	-.07	.01	.01
3.0	-.16	.03	.01
4.0	-.25	-.14	.07
6.0	-.10	-.06	.15

CHAPTER IV DISCUSSION

Interpretation of Results: Pre-Exposure Measures

Acoustic Reflex Threshold

Acoustic reflex threshold can be influenced by many factors. Variables known to affect threshold determination include the nature of the activating stimulus, the sensitivity of the equipment used to monitor AR activity, and the method used to analyze the response. In this study, acoustic reflex thresholds were established for two different broad spectrum signals (broadband noise and industrial noise). Mean threshold values for the broadband and industrial noise elicitors were 72.4 and 73.7 dB SPL, respectively. These values are in good agreement with the literature. Data obtained in other laboratories have reported broadband threshold values between 71 and 77 dB SPL for similar populations (Silman & Gelfand, 1979; Wilson, 1981; Silman, 1979). Of particular importance was the fact that AR thresholds for the two activators were essentially the same. Previous investigations show that AR threshold is highly dependent on bandwidth and spectral density of the

eliciting signal (Peterson & Liden, 1972; Popelka, Margolis & Wiley, 1976). Equal threshold values for the two elicitor conditions would seem to indicate that the spectral content of the two stimuli were similar. These findings suggest that although slight differences were observed in the FFT analysis of the broadband and industrial noise signals (see Figure 7), those differences were not great enough to influence AR threshold sensitivity.

Acoustic Reflex Magnitude

Procedural differences such as stimulus intensity level, activator frequency and mode of presentation have all been reported to cause changes in AR magnitude values. Even under identical stimulus conditions, many investigators still report large inter-subject variability (Dallos, 1964, Silman & Gelfand, 1981; Hepler, 1984; Moul, 1985). Wilson (1981) showed that mean impedance change for a 220 Hz activating signal presented at supra-threshold levels (18 dB SL re: AR threshold) was 800 ohms, with standard deviations as great as 550 ohms. This study also found a great deal of variability in AR magnitude across subjects as demonstrated in Figure 18. Group mean data, however, were similar to those reported in the literature. Wilson (1979) and Moul (1985) reported average acoustic admittance

values (Y_a) of approximately .100 mmhos for a broadband elicitor presented at 16 dB SL (re: AR threshold). This value is in excellent agreement with the .101 mmho value obtained for subjects in this study. Another interesting finding was the consistent behavior demonstrated for the two different elicitor conditions. Subjects demonstrating larger magnitude values for the broadband noise elicitor also showed large magnitude values for the industrial noise signal.

As expected, there was a general increase in AR magnitude per unit increase in stimulus presentation level. The range of magnitude values recorded in this investigation (.01 to 1.01 mmhos) are consistent with those reported in the literature (Wilson & McBride, 1978; Hepler, 1984; Moul, 1985). Wilson and McBride (1978) showed approximately a .0054 mmhos/dB change in admittance which compared favorably to the .0055 mmho/dB change found in this study. The complete dynamic range of AR magnitude could not be evaluated due to limitations of the equipment (106 dB SPL) and subject discomfort. Research using similar activating signals (broadband noise), have reported dynamic range values between 34 dB to greater than 50 dB (Silman, Popelka, & Gelfand, 1978; Wilson & McBride, 1978; Wilson, 1981).

Acoustic Reflex Latency

Measurement of AR latency depends on many factors including characteristics of the eliciting stimulus, instrument response time, and mode of presentation. Latency values are also influenced by the operational definitions used to quantify AR temporal properties. Borg (1972) and Moller (1972) defined onset latency as the time between stimulus onset and the point where the AR reaches 10% of its maximum response amplitude. Colletti (1975) utilizes 5% of maximum immittance change as the definition of onset latency. Other investigators suggest that any measurable immittance change from baseline activity could be used to define onset latency (Metz, 1951; Sunderland, 1974).

The present study used four separate latency values to evaluate the temporal characteristics of the acoustic reflex system. The first latency value (L1) was defined as the time in msec from the beginning of AR response to 10% of the maximum steady state admittance change. Previous studies indicate that onset properties of the AR are directly related to certain parameters of the eliciting signal (Dallos, 1964; McPherson & Thompson, 1977). Onset latency, for example, is inversely related to stimulus intensity level. The greater the intensity of the activator, the shorter the onset latency of the response (Borg, 1972;

Bostra, Russolo, & Silverman, 1984). Mean latency values for this study at AR threshold were 420 msec and declined to 240 msec at supra-threshold levels (16 dB SL re: AR threshold). These findings are in good agreement with values reported by Moul (1985) using a similar definition of AR onset latency. However, they are considerably longer than data presented by Moller (1984), who reported average onset times between 150-250 msec. This disparity is most likely due to procedural differences in quantifying onset latency of the AR response. In this study, threshold determination and latency values were evaluated with the help of computer programs specifically designed to measure AR activity. Such procedures are undoubtedly more sensitive to changes in AR activity than the visual inspection method used in some of the earlier studies. Subsequently, a higher number of acoustic reflex waveforms with very gradual rise times were included in this data pool. This resulted in longer onset latency values than usually described in the literature.

The second latency measure, L2, defined as the time in msec from the beginning of the AR response to 90% of the maximum steady state admittance change, was found to be a static measure of AR latency. Latency values changed very little across stimulus presentation level (see Figure 20). The entire range of measurements occurred in less than 40 msec. This finding shows that stimulus intensity level has virtually no affect on L2 latency values.

A third factor that is derived from L1 and L2 is rate of change in admittance during stimulus onset (Slope 1). This measure was calculated as the change in mmhos per msec during initial stimulus presentation. Findings show that as intensity level increases, so does the rate of change in the AR response. Values obtained in this study ranged from .00004 mmhos/msec at threshold, to .00020 mmho/msec at supra-threshold levels (16 dB SL re: AR threshold). The onset response of the acoustic reflex can now be described as follows. Regardless of stimulus intensity level, the time to reach maximum response amplitude (L2), requires approxiamtely 650-700 msec. As elicitor intensity level increases, initial onset of AR activity (L1) decreases. Simultaneously, the rate of change in admittance (S1) increases, to compensate for the larger admittance changes observed at supra-threshold levels. These findings suggest that AR onset characteristics are principally the result of an interaction of changes occurring in L1 and S1 properties, while L2 values remain relatively constant.

It has been reported that offset latency is less dependent on stimulus parameters than AR onset latency. McPherson and Thompson (1977) suggested this nonlinearity may be due to inherent differences in the process of contraction vs. relaxation of the AR response, and therefore, should be considered as two separate events of AR activity.

Borg (1976) suggests that offset properties may represent a closer estimate of neural conduction time of the AR system. He states that following cessation of the eliciting signal, the change in muscle tension will immediately be present in the AR response, since no slackness of the system has to be overcome as during AR onset. Borg (1976) cautions, however, that any latency measure obtained by immittance equipment only estimates neural transport time. The values obtained by such procedures invariably include mechanical delays of the muscle and middle ear system.

Relaxation properties of the AR response were evaluated using latency measures L3 and L4. These values have been described as the time from the termination of the initial admittance change to 90% and 10% of the steady state admittance value. In general, L3 values were less dependent on stimulus parameters than L1, but did demonstrate an inverse relationship to stimulus intensity level. Average L3 values near threshold were approximately 250 msec, decreasing to 190 msec at 16 dB SL (re: AR threshold). Notice the range of latency values for this measure (60 msec) is considerably less than the over 200 msec range obtained for onset latency values (L1). This finding would seem to support previous research that indicates offset properties of the acoustic reflex system are less dependent on parameters of the eliciting signal (McPherson & Thompson, 1977; Borg, 1976). The change in L3 latency across stimulus

presentation level can be explained by reviewing basic neural physiology.

Borg (1976) speculates that the decrease in offset latencies typically observed at low and moderate intensity levels may be the result of an orderly arrangement of fast and slow motor units in the AR system. Slow units with lower thresholds generally exhibit slower relaxation times than faster units with higher thresholds. Therefore, it seems possible that as stimulus level increases, a greater number of higher threshold, faster conduction fibers are participating in the AR relaxation process. This may explain why offset values show a slight decrease in absolute latency with increases in stimulus presentation level.

L4 values on the other hand showed an increase in latency per unit increase in stimulus intensity level. This is due to the operational procedures used to define L4. As stimulus intensity increases, so does the magnitude of the observed admittance change. Because L4 represents the time required by the AR system to return to its quiescent state, larger magnitude values require longer periods of time to achieve complete relaxation. Therefore, L4 values will naturally be longer at supra-threshold levels.

Adaptation

Much of the existing literature describing AR adaptation involves the use of constant pure tone or noise elicitors (Dallos, 1964; Johansson, Kylin, & Langfy, 1967; Anderson, Barr, & Wedenberg, 1969). It has been shown that AR magnitude declines quite rapidly during elicitation by signals with fixed intensity and spectral content. Other studies have demonstrated that AR activity can be sustained with short pauses, or changes in spectral characteristics of the eliciting signal (Borg & Odman, 1979; Hetu & Careau, 1977; Gjaevenes & Sohoel, 1966). Apparently, the dynamic properties of the acoustic reflex system vary in response to changes in the fatiguing stimulus. Two acoustic features that appear to be intimately related to these changes are temporal properties and frequency content of the signal. In view of these findings, this study choose to investigate adaptation properties of continuous and temporally changing AR elicitors.

Adaptation measures were collected for three broad spectrum signals displaying significantly different temporal qualities. Measures for continuous broadband noise (standard), industrial noise, and interrupted broadband noise (RIT) were obtained during the pre-exposure session. Adaptation was calculated from the difference between AR onset magnitude vs. offset magnitude, and expressed as percent adaptation. The continuous broadband signal

displayed the greatest decline in magnitude (77%), which is in good agreement with the findings of previous investigators. Hepler (1984) and Moul (1985) reported percent adaptation values of 83% and 85%, respectively in their studies of normal hearing subjects. Less adaptation resulted when short pauses were introduced in the sustained broadband signal. Percent adaptation for the RIT procedure was 61%, which was less than the adaptation value obtained for the continuous broadband signal. This would seem to support the work of previous studies that report less adaptation when changes in temporal characteristics of the fatiguing stimulus are present (Hetu & Careau, 1977). Acoustic reflex behavior in response to industrial noise stimulation was also evaluated.

Research would seem to indicate that if the fatiguing stimulus changes in temporal and frequency content, as it does in most industrial situations, resistance to the adaptation process should be even greater than for the other elicitor conditions (Borg & Odman, 1979; Hetu & Careau, 1977; Gjaevenes & Sohoel, 1966). Findings showed that this assumption was correct. Greatest resistance to the process of adaptation was demonstrated by the acoustic reflex system in response to industrial noise stimulation. Values for this measure were consistently less across individuals, resulting in a mean percent adaptation score of only 35%. These results appear to support other

studies that indicate whenever changes of intensity and frequency occur in the fatiguing signal, AR adaptation is greatly reduced. Some investigators believe that the AR undergoes a series of relaxation responses followed by reactivation (Hetu & Careau, 1977). Due to this action, the AR is able to maintain tension on the ossicle chain for longer periods of times without significant adaptation. In light of this evidence, it appears that the acoustic reflex may play a more important role in industrial environments than previously anticipated.

Acoustic reflex behavior in response to interrupted signals was further evaluated by examining individual response magnitude (R1 and R2) for the three RIT conditions. Results suggest that the longer the interruption interval, the greater the magnitude of the RIT response. This finding seems appropriate since the longer interruption intervals allow more time for potential changes in admittance to occur. Close examination of response magnitudes for the 500 and 750 msec RIT procedures reveal similar values for the two elicitor conditions. Examination of L4 values may provide additional information as to the reason for this finding.

Recall that L4 represents the time required by the AR system to return to 90% of its quiescent state. Examination of L4 values for this group of subjects reveal latencies of approximately 500 msec when stimulated at

16 dB SL (re: AR threshold). This means that if a sustained signal were interrupted for a duration of at least 500 msec, it would have sufficient time to return to its quiescent state. In other words, all of the potential change in admittance that would occur did occur within the first 500 msec following cessation of the eliciting signal. This 500 msec time interval, therefore, appears to represent the nominal value required by the AR system to approach maximum offset amplitude.

Another interesting finding was encountered upon comparison R1 and R2 values. By definition, R1 magnitude is the first phase of the RIT response, and represents the change in middle ear admittance following cessation of the eliciting signal. R2 results from the re-elicitation of AR activity due to the re-introduction of the broadband signal. Comparisons of each phase of RIT behavior revealed that R2 (re-elicitation) magnitude was consistently greater than R1 (offset) magnitude. One possible explanation for this finding may be that even short pauses in a fatiguing stimulus allow partial recovery of receptor cells and neural elements along the cochlear partition. Since the activating signal in this experiment was presented at supra-threshold levels, the entire cochlear partition could have been involved in this recovery process. This would allow greater participation from a higher number of receptor cells generating more neural activity upon

re-introduction of the eliciting signal. This increased neural energy would translate into vigorous contraction of the stapedial muscle which would result in greater R2 magnitude.

Changes in RIT magnitude over time were also evaluated in this study. Results show that RIT magnitude declines quite rapidly. Half life values for the three RIT procedures was less than one minute for all stimulus conditions. Again, the length of the interruption interval played a crucial role in the adaptation process. The 250 msec RIT declined most rapidly, followed by the 500 and 750 msec RIT procedures. These findings appear to indicate that as total exposure time to the fatiguing stimulus decreases, resistance to the process of adaptation increases.

Effects of the Industrial Noise Exposure

Behavioral Threshold Shift

As expected, the industrial noise exposure created significant threshold shift at several different frequencies. Greatest shifts in hearing sensitivity occurred in the 2.0 to 6.0 kHz region. The degree of hearing loss created in this study was similar to values obtained by other investigators (Hepler, 1984; Mills, Adkins, & Gilbert, 1981).

One somewhat surprising result was the frequency at which maximum TTS occurred. Data show that 23 of the 30 subjects suffered greatest temporary threshold shift at 3.0 kHz. Similar exposures to broad spectrum stimuli produce greatest threshold shift at 4.0 kHz. Tonndorf (1976) suggested this is primarily the result of resonance properties of the outer ear and the inherent filtering of low frequency energy provided by the middle ear system. Schuknecht (1974) postulates that the Organ of Corti is uniquely vulnerable to excess mechanical stimulation in the 4.0 kHz region of the cochlear partition. While these findings support the general pattern of TTS in the 3.0-4.0 kHz region, results do not explain why the greatest threshold shift was suffered by most of the subjects at 3.0 kHz.

One of the main goals of this study was to investigate auditory behavior in response to an industrial environment. Great effort was made to obtain a "typical" industrial noise sample with broad, flat frequency response. The nature of this task did not allow total control over spectral content of the fatiguing stimulus. Recall that the industrial exposure consisted primarily of noise created by a stone grinder with background shop activity. Although FFT analysis showed the signal to be relatively flat from 1.0 to 6.0 kHz, additional acoustic energy was present in the lower frequencies. Figure 7 demonstrates that the

industrial noise sample was indeed similar to a broadband signal. Close inspection of the FFT graph, however, reveals an additional 5 dB of acoustic energy at 1.5 kHz. Research with octave band exposures of human subjects indicates that maximum TTS occurs one-half to one octave above the exposure frequency (Davis, Morgan, Hawkins, Galambos, & Smith, 1950; Hirsh & Bilger, 1955; Melnick, 1978). It, therefore, seems possible that the 5 dB peak at 1.5 kHz was significant enough to shift the point of maximum TTS from 4.0 to 3.0 kHz. A second possibility to consider was the role that ear canal volume may have played in the development of TTS. Hepler (1984) was able to demonstrate a significant relationship between TTS and estimates of ear canal volume following broadband noise exposure. He reported that subjects with larger than average ear canal volume suffered greatest TTS at 3.0 kHz, while subjects with smaller ear canal volumes experienced greatest TTS at 6.0 kHz. This does not appear to be the case in this experiment since there was no relationship demonstrated between volume and TTS at any test frequency.

Individual values of maximum and minimum TTS at particular test frequencies varied dramatically. Threshold shifts resulting from the noise exposure were as large as 31.5 dB and as little as 0 dB. In no case did maximum TTS values approach the 40 dB level. Research indicates that this may be the point where permanent threshold shift occurs

(Ward, Glorig, & Sklar, 1959). As presented in Table 3, recovery from the temporary hearing loss was complete following 24 hours of quiet.

Acoustic Reflex Threshold

Data obtained from this study reveals that approximately 5 dB of AR threshold shift resulted from the two hour industrial noise exposure. While individual values for reflex threshold shift (RTS) varied considerably (-4 to 16 dB), average RTS values were similar to those obtained by other investigators. Borg, Nilsson and Liden (1979) reported 4 dB of RTS in a group of industrial workers whose AR thresholds were measured before and after a typical workday in a shipyard. Gerhardt, Melnick and Ferraro (1979) demonstrated approximately 4 dB of RTS (in a group of chinchillas) following the first two hours of an eight hour noise exposure. Nilsson, Borg and Liden (1980) also reported approximately 4 dB of reflex threshold shift in humans following noise exposure. These studies appear to confirm that AR threshold represents another component of the auditory system that is influenced by exposure to noise.

The etiology of RTS following noise exposure is still under evaluation. Engstrom and Borg (1983) believe that RTS may be related to inner hair cell changes along

the Organ of Corti. These authors were able to demonstrate that AR threshold shift occurred in a group of rabbits exhibiting lesions of inner hair cell stereocilia (Borg & Engstrom, 1982). Good correlation between extent of injury to IHC stereocilia, and acoustic reflex threshold was also observed (Engstrom & Borg, 1981; Borg & Engstrom, 1982). Liberman and Mulroy (1982) also found changes in IHC stereocilia in cats following chronic noise exposure. They went on to report that IHC stereocilia in their group of animals were the structures most susceptible to the effects of noise. While such evidence is far from conclusive, it does suggest that elevation in AR threshold may be linked to alterations of inner hair cell structures. Further study is required before a definitive answer to this question is obtained.

Acoustic Reflex Magnitude

Significant changes in acoustic reflex magnitude were not obtained following noise exposure. These findings are in direct contrast to the work of Gerhardt and Hepler (1983), who demonstrated a reduction in AR magnitude following a 1.0 kHz exposure of four hours. This apparent contradiction may be explained by procedural differences between studies. Pure tone elicitors were used in their study (0.5, 1.4, 2.0 kHz) in contrast to the broadband noise

elicitor implemented in this experiment. The noise exposure was also considerably different (1.0 kHz octave band vs. industrial noise). One possible explanation of this apparent discrepancy may be related to the tonotopic organization of the cochlear partition.

AR elicitation by pure tone stimuli is most dependent on the integrity of sensory and neural units within a limited area of the cochlea. Although contributions from distal portions of the cochlea may be involved in the response, they are not as crucial to normal sensory function as structures near the site of maximum stimulation. The 1.0 kHz exposure used by Gerhardt and Hepler (1983) created greatest TTS and shifts in AR magnitude at 1.4 and 2.0 kHz. This is the area along the basilar membrane maximally affected by the 1.0 kHz exposure. If changes in AR behavior were to occur, it would undoubtedly be at these frequencies. Broadband elicitors, however, are not as dependent on a limited area of the cochlea for normal sensory function. Contributions from much larger areas of the cochlea are involved when the auditory system is stimulated by a broadband signal. Consequently, the potential to compensate for decreased sensitivity of a limited number of sensory structures is greater under broadband elicitor conditions. It is possible that differences observed between this study and previous investigations of AR magnitude following noise exposure

may be explained by the choice of elicitor used to sample AR behavior.

Acoustic Reflex Latency

Onset properties of the acoustic reflex are highly dependent on parameters of the eliciting signal. McPherson and Thompson (1977) suggest the AR onset response is actually an energy related phenomenon. Stimulus rise-time and intensity affect the total energy available to the system, and dictate AR onset behavior. Offset latency, on the other hand, is relatively independent of stimulus parameters and represents the system's response to cessation of the eliciting signal. Thus, offset latency is directly related to the integrity of both sensory structures in the cochlea and neural units in the auditory pathways.

Onset and offset properties of the acoustic reflex were affected differently by the industrial noise exposure. Onset responses (L1 and L2) remained unchanged following noise exposure, while offset latencies (L3 and L4) revealed significant delays for post-exposure measures. It is possible that onset latencies were not delayed due to their intimate relationship with parameters of the eliciting signal. Any change in AR function caused by the industrial noise may have been insignificant compared to the influence

of stimulus characteristics. In contrast, offset properties reflect conditions of the sensory and neural structures in the system and have an increased potential to be disrupted during conditions of TTS. The etiology of this behavior is controversial.

Norris, Stelmachowicz, and Taylor (1974) reported that subjects with sensori-neural hearing loss exhibited longer offset latencies than subjects with normal hearing. They theorized that differences between the two groups resulted from changes in cochlear function of the hearing impaired subjects. In contrast, Borg (1976) reported longer offset latencies in animals with lesions of the auditory brainstem. He suggested that the group of hearing-impaired subjects used in the previous study presented concomitant retro-cochlear involvement. By creating temporary hearing loss in normal hearing subjects, this experiment offers a unique model of cochlear stress while controlling for the possible contaminating affects of retro-cochlear involvement.

Although changes in central auditory function may occur after long term noise exposure that results in permanent hearing loss, there is no evidence to suggest this would happen under conditions of TTS. If delayed offset latencies are the result of retro-cochlear rather than cochlear involvement as suggested by Borg, then offset latencies should not be prolonged when normal hearing subjects experience TTS. This was not the case. Offset

latencies were consistently longer in this group of subjects experiencing temporary cochlear hearing loss. This evidence would seem to support the work of Norris, Stelmachowicz, and Taylor (1974) who reported that longer offset latencies are attributable to changes in the peripheral rather than central auditory system.

Adaptation

Post-exposure data revealed a general trend toward an increase in percent adaptation following industrial noise exposure. This finding was evident for RIT procedures as well as standard measures of adaptation. However, statistically significant changes were only obtained for RIT elicitation conditions. This difference cannot be easily explained, but may be due to the small sample size used in this study and the variability observed in these measures. Perhaps different measurement techniques, or a larger sample size would have revealed a significant change following noise exposure for both experimental conditions.

It was anticipated that analysis of individual RIT responses would also demonstrate change following industrial noise exposure. Again, this was not the case. Post-exposure estimates of R1 and R2 magnitude across all three RIT conditions revealed no significant change following

noise exposure. One possible explanation for this finding would be the obvious difference in RIT quantification between measures of percent adaptation and RIT magnitude. Percent adaptation values were determined by calculating the change from onset magnitude to offset magnitude during the experimental sampling period. RIT magnitude values on the other hand, were obtained by measuring offset and re-elicitation characteristics of the response every 7.5 seconds. By interrupting a sustained broadband signal, the RIT is creating a complex situation between stimulus parameters and AR behavior. This interaction is superimposed on the gradual decline in AR magnitude typically seen in standard measures of acoustic reflex adaptation. These procedural differences in the analysis of the RIT data may have contributed to this apparent discrepancy although other explanations may exist.

Correlations

Potential relationships between measures of acoustic reflex behavior and temporary threshold shift failed to demonstrate that AR activity can be extremely useful in explaining the variability encountered in TTS. Correlational analysis between AR threshold, AR threshold shift, and AR adaptation failed to demonstrate significant

correlations to TTS created at any test frequency. Significant correlations were obtained between acoustic reflex magnitude and offset latencies.

Of all the measures of AR activity, magnitude provided the most predictive information about temporary threshold shift. AR magnitude demonstrated the strongest relationship to TTS in the frequencies experiencing greatest threshold shift (3.0 and 4.0 kHz). This relationship was consistently negative, indicating subjects with greater AR magnitude suffered less TTS following industrial noise exposure (average correlation $r = -.45$). These findings support previous investigations. Gerhardt and Hepler (1983), utilized an average AR magnitude value (6-10 dB SL) to predict TTS following a 1.0 kHz octave band noise exposure. Results indicate that a significant negative correlation ($r = -.75$) was present between AR magnitude and TTS at 1.4 kHz. In addition, Hepler (1984) found consistent negative correlations between expressions of TTS created by broadband noise exposure, and AR magnitude elicited by broadband activators (6-12 dB SL). Moul (1985), reported similar results utilizing 0.5 kHz octave band noise and a broadband elicitor. His data support the contention that temporary threshold shift and AR magnitude are inversely related. These findings appear to suggest that the relationship between AR magnitude and expressions of TTS provide useful information about the variability exhibited in behavioral threshold shift.

The concept of shorter AR onset latency providing greater protection from industrial noise was not supported in this study. No predictive value was obtained for correlations between AR onset parameters and expressions of TTS. A pattern of significant correlations were obtained for offset latency L4. Findings reveal that a significant relationship exists between measures of L4 and TTS created at 4.0 kHz. Similar correlations were not obtained for other test frequencies. This suggests that offset latency may represent another variable in the development of temporary threshold shift.

In general, measures of acoustic reflex behavior do not appear to be extremely useful in predicting temporary threshold shift. AR magnitude accounts for some of the variability in TTS measures, as does offset latency L4.

Summary

Several changes in auditory behavior were noted following the two-hour industrial exposure to 90 dB SPL. As expected, shifts in behavioral thresholds at octave and half-octave intervals were obtained in the 3.0-6.0 kHz region. One somewhat surprising result was the fact that maximum TTS occurred at 3.0 kHz instead of 4.0 kHz.

This was the result of increased acoustic energy in the 1.5 kHz region of the industrial noise sample. The noise exposure also produced significant changes in measures of acoustic reflex threshold, acoustic reflex offset latencies and measures of acoustic reflex adaptation.

This study was designed to determine if differences exist in percent adaptation for the three elicitors evaluated in the pre-exposure session (continuous broadband noise, industrial noise, and interrupted broadband noise). Results show that greatest adaptation occurred for the continuous broadband noise followed by the interrupted broadband noise. The least adaptation occurred for the industrial noise signal. These findings are the result of the temporally changing nature of industrial noise which increases the resistance to the adaptation process.

Finally, measures of acoustic reflex activity were correlated to expressions of TTS in an effort to explain part of the variability encountered in studies of auditory fatigue. Only two measures of AR behavior demonstrated statistically significant relationships to TTS. Acoustic reflex magnitude showed a strong negative correlation to TTS at the frequencies most affected by the noise exposure (3.0 and 4.0 kHz). These findings indicate that the larger the magnitude of the AR response, the less the resultant TTS. Offset latency (L4) also showed a significant negative correlation to TTS at 4.0 kHz. In general,

however, the results failed to demonstrate a strong relationship between acoustic reflex activity and expressions of TTS.

REFERENCES

- American National Standards Institute (ANSI), 1960. Acoustic terminology, 51.1. New York: American National Standards Institute.
- American National Standards Institute (ANSI), 1969. Specifications for audiometers, 53.6. New York: American National Standards Institute.
- Anderson, H., Barr, B., & Wedenberg, E., 1969. Intra-aural reflexes in retro-cochlear lesion. In C. A. Hamberger & J. Wersall (Eds.), Nobel Symposium Disorders of the skull base region. Stockholm: Almquist & Wekell.
- Anderson, H., Barr, B., & Wedenberg, E., 1970. Early diagnosis of VIII nerve tumors by acoustic reflex test. Acta Oto-Laryngol., 263, 232-237.
- Antablin, J., Lilly, D., & Wilson, R., 1980. Intra-subject variability of acoustic reflex adaptation in normal listeners. American Speech-Language-Hearing Association Convention. Detroit.
- Barry, S.J., & Resnick, S.B., 1976. Comparison of acoustic reflex and behavioral thresholds as a function of stimulus frequency and duration. J. Am. Audiol. Soc., 2, 35-37.
- Bennett, M., 1984. Impedance concepts relating to the acoustic reflex. In S. Silma (Ed.), The acoustic reflex: Basic principles and clinical applications. Orlando, FL: Academic Press.
- Block, M.G., & Wiley, T.L., 1979. Tutorial: Static acoustic-immittance measurements. J. Speech Hearing Res., 22, 677-696.
- Borg, E., 1972. On the change in acoustic impedance of the middle ear as a measure of middle ear reflex activity. Acta Oto-Laryngol., 74, 163-171.

- Borg, E., 1973. On the neuronal organization of the acoustic middle ear reflex. A physiological and anatomical study. Brain Res., 49, 101-23.
- Borg, E., 1976. Dynamic characteristics of the intra-aural muscle reflex. In A. Feldman & L. A. Wilber (Eds.), Acoustic impedance and admittance: The measurement of middle ear function. Baltimore, MD: Williams and Wilkins Company.
- Borg, E., 1977. The intra-aural muscle reflex in retro-cochlear pathology: A model study in the rabbit. Audiology, 16, 316-330.
- Borg, E., Counter, S.A., & Rosler, G., 1984. Theories of middle-ear muscle function. In S. Silman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.
- Borg, E., & Engstrom, B., 1982. Acoustic reflex after experimental lesions to inner and outer hair cells. Hearing Res., 6, 25-34.
- Borg, E., Nilsson, R., & Engstrom, B., 1983. Effect of the acoustic reflex on inner ear damage induced by industrial noise. Acta Oto-Laryngol., 96, 361-369.
- Borg, E., Nilsson, R., & Liden, G., 1979. Fatigue and recovery of the human acoustic reflex in industrial noise. J. Acoust. Soc. Am., 65, 846-848.
- Borg, E., & Odman, B., 1979. Decay and recovery of the acoustic stapedius reflex in humans. Acta Oto-Laryngol., 87, 421-428.
- Bosatra, A., Russolo, M., & Silverman, C.A., 1984. Acoustic reflex latency: State of the art. In S. Silman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.
- Carder, H., & Miller, J., 1972. Temporary threshold shifts from prolonged exposure to noise. J. Speech Hearing Res., 15, 603-623.
- Chermak, G.D., Dengerink, J.E., & Dengerink, H.A., 1983. Test-retest reliability of the acoustic stapedius reflex. Audiology, 22, 136-143.
- Chun, C.D., & Raffin, M.J.M., 1979. Reliability of acoustic-reflex threshold measurements. Paper presented to the annual meeting of American Speech-Language-Hearing Association, Atlanta, Georgia.

- Colletti, V., 1975. Some stapedius reflex abnormalities in multiple sclerosis. Audiology, 14, 63-71.
- Dallos, P.J., 1964. Dynamics of the acoustic reflex: Phenomenological aspects. J. Acoust. Soc. Am., 36, 2175-2183.
- Dallos, P.J., 1973. The auditory periphery: Biophysics and physiology. New York: Academic Press, Inc.
- Davis, H., 1958. ANEHIN: Auditory and non-auditory effects of high intensity noise. Central Institute for the Deaf and U.S. Naval School of Aviation Medicine, Pensacola, Florida, Joint Project report No. 7.
- Davis, H., Morgan, C.T., Hawkins, J.E., Galambos, R., & Smith, F.W., 1950. Temporary deafness following exposure to loud tones and noise. Acta Oto-Laryngol., Suppl. 88, 1-24.
- Djupesland, G., 1967. Contraction of the tympanic muscles in man. Thesis, Universitetsforlaget, Oslo, Norway.
- Djupesland, G., Flottorp, G., & Winther, F.O., 1966. Size and duration of the acoustically elicited impedance change in man. Acta Oto-Laryngol., 224, 220-228.
- Djupesland, G., Flottorp, G., & Winther, F.O., 1967. Size and duration of acoustically elicited impedance change in man. Acta Oto-Laryngol., Suppl. 224, 220-227.
- Djupesland, G., & Zwislocki, J.J., 1971. Effect of temporal summation on the stapedius reflex. Acta Oto-Laryngol., 71, 262-265.
- Djupesland, G., & Zwislocki, J.J., 1973. On the critical band in the acoustic stapedius reflex. J. Acoust. Soc. Am., 54, 1157-1159.
- Dougherty, J.D., 1970. Stress-induced disease. In Human response to sonic booms. FAA Report No. 70-2. Washington, DC: Dept. of Transportation.
- DuVerney, G.J., 1683. Traite de l'organe de l'ouie. Paris: Estienne Michallet.
- Engstrom, B., & Borg, E., 1981. Lesions to cochlear inner hair cells induced by noise. Arch. Otorhino-laryngol., 230, 279-284.

- Engstrom, B., & Borg, E., 1983. Cochlear morphology in relation to loss of behavioral, electrophysiological, and middle ear reflex thresholds after exposure to noise. Acta. Oto-Laryngologica, Suppl. 402.
- Fabricius ab Agapendente, Hieronymus, 1600. De visione, voce et auditu. Venice.
- Feldman, A.S., 1976. Tympanometry: Procedures, interpretations and variables. In A. S. Feldman & L. A. Wilbur (Eds.), Acoustic impedance and admittance: The measurement of middle-ear function. Baltimore, MD: Williams and Wilkins Company.
- Feldman, A.S., & Wilbur, L.A., 1976. Introduction. In A. S. Feldman & L. A. Wilbur (Eds.), Acoustic impedance and admittance: The measurement of middle ear function. Baltimore, MD: Williams and Wilkins Company.
- Fletcher, J.L., & King, W.P., 1963. Susceptability of stapedectomized patients to noise and induced temporary threshold shifts. Ann. Otol. Rhinol. Laryngol., 72, 900-907.
- Flottorp, G., Djupesland, G., & Winther, F., 1971. Acoustic stapedius reflex in relation to critical bandwidth. J. Acoust. Soc. Am., 49, 457-465.
- Forquer, B.D., 1979. The stability of and the relationship between the acoustic reflex and uncomfortable loudness levels. J. Am. Aud. Soc., 5, 55-59.
- Fowler, C.G., & Wilson, R.H., 1984. Adaptation of the acoustic reflex. Ear & Hearing, 5, 281-288.
- Gans, D.P., Sweetman, R.H., & Carlson, H.C., 1972. Use of high speed photography in analysis of the acoustic reflex. J. Acoust. Soc. Am., 51, 1826-1827.
- Gelfand, S.A., 1984. The contralateral acoustic-reflex threshold. In S. Silman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.
- Gelfand, S.A., Silman, S., & Silverman, C.A., 1981. Temporal summation in acoustic reflex growth functions. Acta Oto-Laryngol., 91, 177-182.
- Gerhardt, K.J., & Hepler, E.L., 1983. Acoustic stapedius reflex and behavioral thresholds following exposure to noise. J. Acoust. Soc. Am., 74, 109-114.

- Gerhardt, K.J., Melnick, W., & Ferraro, J.A., 1979. Reflex threshold shift in chinchillas following a prolonged noise exposure. J. Speech Hearing Res., 22, 63-72.
- Gjaevenes, K., & Sohoel, T.H., 1966. Reactivating the acoustic stapedius muscle reflex by adding a second tone. Acta Oto-Laryngol., 62, 213-216.
- Hepler, E.L., 1984. Acoustic-reflex dynamics and temporary threshold shift. Doctoral Dissertation, University of Florida.
- Hetu, R., & Careau, P.U., 1977. Decay of the acoustic reflex during steady-state and intermittent noise exposures. Paper presented at 93rd meeting of the Acoustical Society of America, University Park, PA.
- Hirsh, I.J., & Bilger, R.C., 1955. Auditory threshold recovery after exposure to pure tones. J. Acoust. Soc. Am., 27, 1186-1190.
- Hirsh, I.J., & Ward, W.D., 1952. Recovery of auditory threshold after strong acoustic stimulation. J. Acoust. Soc. Am., 24, 131-141.
- Holmes, A.E., 1978. An investigation of the relationship between temporary threshold shift and the acoustic reflex. Master's Thesis, Idaho State University.
- Jepsen, O., 1963. Middle-ear muscle reflexes in man. In J. Jerger (Ed.), Modern developments in audiology. New York: Academic Press, Inc.
- Jerger, J., 1970. Clinical experience with impedance audiometry. Archives of Otolaryngol., 92, 311-324.
- Jerger, J., Anthony, L., Jerger, S., & Mouldin, L., 1974. Studies in impedance audiometry III. Middle ear disorders. Archives of Otolaryngol., 99, 163-171.
- Jerger, J., Jerger, S., & Mauldin, L., 1972. Studies in impedance audiometry: Normal vs. sensori-neural ears. Archives of Otolaryngol., 96, 513-523.
- Jerger, J., Jerger, S., & Mauldin, L., 1974. Studies in impedance audiometry. Archives of Otolaryngol., 99, 1-9.
- Johansson, B., Kylin, B., & Langfy, M., 1967. Acoustic reflex as a test of individual susceptibility to noise. Acta Oto-Laryngol., 64, 256-262.

- Johnsen, N.J., & Terkildsen, K., 1980. The normal middle ear reflex thresholds towards white noise and acoustic clicks in young adults. Scandinavian Audiology, 9, 131-135.
- Karlovich, R.S., Luterman, B.F., & Abbs, M.H., 1972. Temporary threshold shift reduction as a function of contralateral noise. J. Speech Hearing Res., 15, 792-799.
- Kato, T., 1913. Zur Physiologie der Binnenmuskeln des Ohres. Pfluger's Archiv fur die gesamte Physiologie des Mehschen und der Tiere, 150, 569-625.
- Kiang, N.Y.-S., 1980. Processing of speech by the auditory nervous system. J. Acoust. Soc. Am., 68, 830-835.
- Kiang, N.Y.-S., Watanabe, T., Thomas, E.C., & Clark, L.F., 1965. Discharge patterns of single fibers in the cat's auditory nerve. Research Monograph No. 35. Cambridge, MA: MIT Press.
- Kryter, K.D., 1970. The effects of noise on man. New York: Academic Press, Inc.
- Kryter, K.D., 1972. Non-auditory effects of environmental noise. Am. J. Public Health, 4, 389-398.
- Liberman, M.C., & Mulroy, M.J., 1982. Acute and chronic effects of acoustic trauma: Cochlear pathology and auditory nerve pathophysiology. In R. P. Hamernik, D. Henderson, & R. Salvi (Eds.), New perspectives on noise induced hearing loss. New York: Raven Press.
- Lilly, D.J., 1984. Evaluation of the response time of acoustic immittance instruments. In S. Sidman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.
- Lucae, A., 1874. Accomodation and Accommodationsstorungen des Ohres. Berlinger Klinische Wochenschrift, 14, 163-165.
- Margolis, R.H., 1981. Appendix A: Fundamentals of acoustic immittance. In G.R. Popelka (Ed.), Hearing assessment with the acoustic reflex. New York: Grune & Stratton.
- Margolis, R.H., Dubno, J.R. & Wilson, R.H., 1980. Acoustic reflex thresholds for noise stimuli. J. Acoustic Soc. Am., 68, 892-895.

- Marks, R.G., 1982. Analyzing research data: The basics of biomedical research methodology. Belmont, CA: Lifetime Learning Publications.
- McPherson, D.L., & Thompson, D., 1977. Quantification of the threshold and latency parameters of the acoustic reflex in humans. Acta Oto-Laryngol., 353, 1-37.
- Melnick, W., 1976. Human asymptotic threshold shift. In D. Henderson, R.P. Hemerik, D.S. Dosanjh, & J.A. Mills (Eds.), Effects of noise on hearing. New York: Raven Press.
- Melnick, W., 1978. Temporary and permanent threshold shift. In D.M. Lipscomb (Ed.), Noise and audiology. Baltimore, MD: University Park Press.
- Melnick, W., & Maves, M., 1974. Asymptotic threshold shift (ATS) in man from 24-hour exposure to continuous noise. Ann. Otol. Rhinol. Laryngol., 83, 820-829.
- Metz, O., 1951. Studies on the contraction of the tympanic muscles as indicated by changes in the impedance of the ear. Acta Oto-Laryng. (Stockh.), 39, 387-405.
- Michael, P.L., & Brenvenue, G.R., 1976. Calibration data for a circumaural headset designed for hearing testing. J. Acoust. Soc. Am., 69, 944-950.
- Miller, J.D., 1958. Temporary hearing loss at 4000 cps as a function of intensity of a three-minute exposure to a noise of uniform spectrum level. Laryngoscope, 68, 660-671.
- Mills, J.H., 1984. Noise-induced hearing loss: State-of-the-art. Paper presented at the American Speech-Language-Hearing Association Convention, San Francisco, CA.
- Mills, J.H., Adkins, W.Y., & Gilbert, R.M., 1981. Temporary threshold shifts produced by wideband noise. J. Acoust. Soc. Am., 70, 390-396.
- Mills, J.H., Gengel, R.W., Watson, C.S., & Miller, J.D. 1970. Temporary changes of the auditory system due to exposure to noise for one or two days. J. Acoust. Soc. Am., 48, 524-530.
- Moller, A.R., 1958. Intra-aural muscle contraction in man, examined by measuring acoustic impedance of the ear. Laryngoscope, 68, 48-62.

- Moller, A.R., 1961. Bilateral contraction of the tympanic muscles in man. Ann. Otol. Rhinol. Laryngol., 79, 735-752.
- Moller, A.R., 1962. Acoustic reflex in man. J. Acoust. Soc. Am., 34, 1532-1534.
- Moller, A.R., 1965. An experimental study of the acoustic impedance of the middle ear and its transmission properties. Acta Oto-Laryng. (Stockh.), 60, 129-149.
- Moller, A.R., 1972. The middle ear. In J.V. Tobias (Ed.), Foundations of modern audiotry theory, vol. 2. New York: Academic Press, Inc.
- Moller, A.R., 1974. The acoustic middle-ear muscle reflex. In W.D. Kiedel & W.D. Neff (Eds.), Handbook of sensory physiology. New York: Springer-Verlag.
- Moller, A.R., 1984. Neurophysiological basis of the acoustic middle ear reflex. In S. Silman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.
- Moul, M.J., 1985. Acoustic-reflex dynamics and temporary threshold shift in octave-band noise exposures. Doctoral Dissertation, University of Florida.
- Nilsson, R., Borg, E., & Liden, G., 1980. Fatigability of the stapedius reflex in industrial noise. Acta Oto-Laryngol., 89, 433-439.
- Norris, T.W., Stelmachowicz, P., Bowling, C., & Taylor, D., 1974. Latency measures of the acoustic reflex, normals vs. sensori-neurals. Audiology, 13, 464-469.
- Norris, T.W., Stelmachowicz, P., & Taylor, D.J., 1974. Acoustic reflex relaxation to identify sensorineural hearing impairment. Archives of Otolaryngology, 99, 194-197.
- Northern, J.L., & Grimes, A.M., 1978. Introduction to acoustic impedance. In J. Katz (Ed.), Handbook of clinical audiology. Baltimore, MD: Williams and Wilkins Company.
- Osterhammel, D., & Osterhammel, P., 1979. Age and sex variations for the normal stapedial reflex thresholds and tympanometric compliance values. Scand. Audiol., 8, 153-158.

- Perlman, H.B., & Chase, T.J., 1939. Latent period of the crossed stapedius reflex in man. Annals of Otology, Rhinology, and Laryngology, 48, 663-675.
- Peterson, J.L., & Liden, G., 1972. Some static characteristics of the stapedial muscle reflex. Audiology, 11, 97-114.
- Pickles, J.D., 1982. An introduction to the physiology of hearing. London: Academic Press, Inc.
- Popelka, G.R., 1984. Acoustic immittance measures: Terminology and instrumentation. Ear & Hearing, 5, 263-267.
- Popelka, G.R., Margolis, R.H., & Wiley, T.L., 1976. Effect of activating signal bandwidth in acoustic reflex thresholds. J. Acoust. Soc. Am., 59, 153-159.
- Richards, A.M., 1975. Threshold of the acoustic stapedius reflex for short-duration tone bursts. J. Audit. Res., 15, 87-94.
- Schuknecht, H.F., 1974. Pathology of the ear. Cambridge, MA: Harvard University Press.
- Selters, W., & Ward, W.D., 1962. Temporary threshold shift with changing duty cycle. J. Acoust. Soc. Am., 34, 122-123.
- Shanks, J., 1979. An evaluation of three procedures for estimating the volumes of human ear canals as a function of ear-canal pressure and a comparison of static acoustic-impedance values from two measurement systems. Doctoral Dissertation, University of Iowa.
- Shanks, J.E., & Lilly, D.J., 1981. An evaluation of the tympanometric estimates of ear canal volume. J. Speech Hearing Res., 24, 557-566.
- Shaw, A.G., 1974. The external ear. In W.D. Keidel & W.D. Neff (Eds.), Handbook of sensory physiology. New York: Springer-Verlag.
- Silman, S., 1979. The effects of aging on the stapedius reflex thresholds. J. Acoust. Soc. Am., 66, 735-738.
- Silman, S., 1984. Magnitude and growth of the acoustic reflex. In S. Silman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.

- Silman, S., & Gelfand, S.A., 1979. Prediction of hearing levels from acoustic reflex thresholds in persons with high frequency hearing losses. J. Speech and Hearing Res., 22, 697-707.
- Silman, S., & Gelfand, S.A., 1981. Effect of sensorineural hearing loss on the stapedius reflex growth function in the elderly. J. Acoust. Soc. Am., 69, 1099-1106.
- Silman, S., Popelka, G.R., & Gelfand, S.A., 1978. The effect of sensorineural hearing loss on acoustic stapedial reflex growth functions. J. Acoust. Soc. Am., 64, 1406-1411.
- Silverman, C.A., Silman, S., & Miller, M.H., 1983. The acoustic reflex threshold in aging ears. J. Acoust. Soc. Am., 73, 248-255.
- Stricker, S., 1880. Studien uber die Sprachvorstellungen. Wien: W. Braumuller.
- Sunderland, E.G., 1974. The effect of increasing stimulus intensity and rise time on the latency period of the acoustic reflex in normal and sensori-neural subjects. Master's Thesis, University of Utah.
- Thompson, D.J., Sills, J.A., Recke, K.S., & Bui, D.M., 1980. Acoustic reflex growth in the aging adult. J. Speech Hearing Res., 23, 405-418.
- Tonndorf, J., 1976. Relationship between the transmission characteristics of the conductive system and noise-induced hearing loss. In D. Henderson, R.P. Hamerik, D.S., Dosanjh, & J.H. Mills (Eds.), Effects of noise on man. New York: Raven Press.
- Tonndorf, J., & Khanna, ., 1968. Submicroscopic displacement amplitudes of the tympanic membrane (cat) measured by a laser interferometer. J. Acoust. Soc. Am., 44, 1546-1554.
- Turner, S.A., 1974. An investigation of the relationship between temporary threshold shift and acoustic impedance. Master's Thesis, Idaho State University.
- von Helmholtz, H., 1868. Die Mechanik der Gehorknochenchen und des Trommelfells. Pfluger's Archiv fur die gesamte Physiologie des Menschen und der Thiere, 1, 1-60.

- Ward, W.D., 1961. Studies on the aural reflex: Contralateral remote masking as an indicator of reflex activity. J. Acoust. Soc. Am., 33, 1034-1045.
- Ward, W.D., 1968. Susceptibility to auditory fatigue. In W.D. Neff (Ed.), Contributions to sensory physiology, vol. 3. New York: Academic Press, Inc.
- Ward, W.D., 1970. Temporary threshold shift and damage-risk criteria for intermittent exposures. J. Acoust. Soc. Am., 48, 561-574.
- Ward, W.D., 1973. Adaptation and fatigue. In J. Jerger (Ed.), Modern developments in audiology, 2nd ed. New York: Academic Press, Inc.
- Ward, W.D., 1979. General auditory effects of noise. In R.W. Cantrell (Ed.), The otolaryngologic clinics of North America: Symposium on noise--its effects and control, vol. 12. Philadelphia, PA: W. B. Saunders Company.
- Ward, W.D., 1980a. Noise-induced hearing damage. In M.M. Paperella & D.A. Shumrick (Eds.), Otolaryngology, Vol. II, 2nd ed. Philadelphia, PA: W. B. Saunders Company.
- Ward, W.D., 1980b. Noise-induced hearing loss: Research since 1973. In J. Tobias, G. Jansen & W.D. Ward (Eds.), Proceedings of the third international congress on noise as a public health problem. Rockville, MD: American Speech-Language-Hearing Association.
- Ward, W.D., Cushing, E.M., & Burns, E.M., 1976. Effective quiet and moderate TTS: Implications for noise exposures standards. J. Acoust. Soc. Am., 59, 160-165.
- Ward, W.D., Glorig, A., & Sklar, D.L., 1958. Dependence of temporary threshold shift at 4 kc on intensity and time. J. Acoust. Soc. Am., 30, 944-950.
- Wiener, F.M., & Ross, D.A., 1946. The pressure distribution in the auditory canal in a progressive sound field. J. Acoust. Soc. Am., 18, 401-408.
- Wiley, T.L., & Karlovich, R.S., 1975. Acoustic reflex response to sustained signals. J. Speech Hearing Res., 18, 148-157.

- Wilson, R.H., 1979. Factors influencing the acoustic immittance characteristics of the acoustic reflex. J. Speech Hearing Res., 22, 480-499.
- Wilson, R.H., 1981. The effects of aging on the magnitude of the acoustic reflex. J. Speech Hearing Res., 24, 406-413.
- Wilson, R.H., & McBride, L.M., 1978. Threshold and growth of the acoustic reflex. J. Acoust. Soc. Am., 63, 147-154.
- Wilson, R.H., Shanks, J.E., Jones, H.C., & Danielson, P.D., 1982. A simplified technique for measuring the temporal characteristics of aural acoustic-immittance instruments. ASHA, 24, 775.
- Wilson, R.H., Shanks, J.E., & Lilly, D.J., 1984. Acoustic reflex adaptation. In S. Silman (Ed.), The acoustic reflex: Basic principles and clinical applications. New York: Academic Press, Inc.
- Wilson, R.H., Steckler, J.F., Jones, H.C., & Margolis, R.H., 1978. Adaptation of the acoustic reflex. J. Acoust. Soc. Am., 64, 782-791.
- Woodford, C., Henderson, R., Hamernik, R., & Feldman, A., 1975. Threshold-duration function of acoustic reflex in man. Audiology, 14, 53-62.
- Zakrisson, J.E., 1974. Experimental studies on the function of the stapedius muscle in man. UMEA University Medical Dissertations, No. 18, Karolinska Institutet, Stockholm, Sweden.
- Zakrisson, J.E., Borg, E., & Blom, S., 1974. The acoustic impedance change as a measure of stapedius muscle activity in man. Acta. Oto., 78, 357-364.
- Zakrisson, J.E., Borg, E., Liden, G., & Nilsson, R., 1980. Stapedius reflex in industrial impact noise: Fatigability and role for temporary threshold shift (TTS). Scandinavian Audiology, Suppl. 12, 326-334.
- Zwislocki, J.J., & Feldman, A.S., 1970. Acoustic impedance of pathological ears. ASHA Monographs No. 15.

BIOGRAPHICAL SKETCH

Gary Philip Rodriguez was born February 13, 1956, in Miami, Florida. His wife, Diane Rodriguez, is also from Miami. They have two children, Casey Philip, and Rebecca Dawn.


Gary received his Bachelor of Science degree in audiology and speech pathology from Florida State University, in June, 1978. In August, 1979, he received a Master of Science degree in audiology, from that same university. Following graduation, he served as a clinical audiologist at Olin E. Teague Veterans Administration Hospital, in Temple, Texas. He then obtained a position as clinical audiologist at Miami Hearing and Speech Center, where he worked for three years before returning to graduate school in August, 1983. He is currently employed at the Florida State University, Department of Communication Disorders, as an assistant professor.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.



Kenneth J. Gerhardt, Ph.D. Chairman
Associate Professor of Speech

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.



Patricia B. Kricos, Ph.D.
Associate Professor of Speech

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.



Alice E. Holmes, Ph.D.
Assistant Professor of Speech

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.



Francis J. Kemker, Ph.D.
Associate Professor of Speech

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Alan Agresti

Alan G. Agresti, Ph.D.
Professor of Statistics

This dissertation was submitted to the Graduate Faculty of the Department of Speech in the College of Liberal Arts and Sciences and to the Graduate School, and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

August, 1986

Dean, Graduate School

UNIVERSITY OF FLORIDA



3 1262 08553 7370